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SOUTHERN MEDICAL ASSOCIATION, Twenty-Fourth Annual Meeting, Louisville, Kentucky, November 12-14, 1930. Pre-Meeting Clinics, November 11, 1930

TABLE OF CONTENTS

A Clinical and Pathological Study of Post-Menopausal Bleeding. Richard W. TeLinde, Baltimore, Md. 571
Discussed by Erasmus H. Kloman, Baltimore, Md.; Willard R. Cooke, Galveston, Tex.

Eggs and Larvae of Common House Fly Found in Human Intestine. Marvin Smith, Miami, Fla. 576
Discussed by J. E. Knighton, Shreveport, La.; W. S. Anderson, Memphis, Tenn.; W. E. Vest, Huntington, W. Va.

The Cystoscopic Appearance of Various Types of Cystoceles. Rex E. Van Duzen, Dallas, Tex. 580
Discussed by H. W. E. Walther, New Orleans, La.; Joseph H. Smith, Memphis, Tenn.; M. M. Coplan, Miami, Fla.; Frank S. Crockett, Lafayette, Ind.

Epiphyseal Separation Due to Muscular Action: Report of Two Cases. Alphonse H. Meyer, Memphis, Tenn. 587
Discussed by Guy A. Caldwell, Shreveport, La.; Donald T. Babcock, Miami, Fla.

Chronic Appendicitis: Diagnosis and Significance. Thomas R. Brown and Ernest H. Gaither, Baltimore, Md. 591
Discussed by Julius Friedenwald, Baltimore, Md.; T. Z. Cason, Jacksonville, Fla.

Sickle Cell (Meniscocytic) Anemia. George S. Graham and Sarah H. McCarty, Birmingham, Ala. 598

Indications for Laminectomy in Spinal Injuries. Charles Edward Dowman, Atlanta, Ga. 607
Discussed by Isidore Cohn, New Orleans, La.; Guy A. Caldwell, Shreveport, La.; Frank P. Strickler, Louisville, Ky.

Osteochondritis Dissecans. Vincent W. Archer and Charles H. Peterson, University, Va. 611
Discussed by Alfred L. Gray, Richmond, Va.; Dean Lewis, Baltimore, Md.; E. H. Teeter, Jacksonville, Fla.

Continued on Next Page

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TABLE OF CONTENTS—Concluded

Acute Pleural Empyema: A Review. Lyle B. West, Chattanooga, Tenn. 615

Diminishing Mortality and Morbidity. Isidore Cohn, New Orleans, La. 620

Discussed by J. Shelton Horsley, Richmond, Va.; I. M. Gage, New Orleans, La.; J. L. Crook, Jackson, Tenn.; J. T. Nix, New Orleans, La.

Thymus. Douglas D. Martin, Tampa, Florida 625

Discussed by J. D. Love, Jacksonville, Fla.; William W. McKibben, Miami, Fla.; H. Leslie Moore, Dallas, Tex.; Chas. E. Conrad, Harrisonburg, Va.

Food Content with Relation to Density and Composition of Stone in Upper Urinary Tract. Owsley Grant and Virgil Simpson, Louisville, Ky. 628

Discussed by A. J. Crowell, Charlotte, N. C.; Neil S. Moore, St. Louis, Mo.; M. M. Coplan, Miami, Fla.; J. Ullman Reaves, Mobile, Ala.

Some Complications of Otitic Sepsis, with Case Reports. Sam B. Marks, Lexington, Ky. 636

Thrombosis of the Sigmoid Sinus and Internal Jugular Vein with Special Reference to the Tobey-Ayers Test. Cuthbert Tunstall, University, Va. 639

Papers of Dr. Marks and Dr. Tunstall discussed by Murdock Equen, Atlanta, Ga.; F. E. Hasty, Nashville, Tenn.; J. W. Downey, Baltimore, Md.; Oscar Wilkinson, Washington, D. C.

Oxygen Therapy in Epilepsy. Pat Murphey, Little Rock, Ark. 647

Discussed by W. E. Gardner, Louisville, Ky.; William Nelson, St. Louis, Mo.; N. R. Smith, Tulsa, Okla.; William A. Smith, Atlanta, Ga.

The Conduct of Labor in Pelvic Deformities. Lester A. Wilson, Charleston, S. C. 653

Discussed by J. Bay Jacobs, Washington, D. C.; James R. McCord, Atlanta, Ga.; Willard R. Cooke, Galveston, Tex.; Mary Freeman, Perrine, Fla.; E. L. King, New Orleans, La.; S. H. Starr, Louisville, Ky.; W. B. Anderson, Nashville, Tenn.; M. C. Wilson, Miami, Fla.

Tree Pollen Hay Fever and Asthma in the South. I. S. Kalin and Emma M. Grothaus, San Antonio, Tex. 658

Discussed by John P. Henry, Memphis, Tenn.; Narcisse Thiberge, New Orleans, La.; Ray M. Balyeat, Oklahoma City, Okla.; H. M. Davison, Atlanta, Ga.

Indications for the Use of Radium in Gynecology. Gerry R. Holden, Jacksonville, Fla. 662

Discussed by Lawrence R. Wharton, Baltimore, Maryland.

Gastric Carcinoma with Esophageal Obstruction. C. Larimore Perry, Miami, Florida 665

Whole Time Medicine. Roy K. Flannagan, Richmond, Va. 666

Discussed by J. A. Ferrell, New York, N. Y.; A. T. McCormack, Louisville, Ky.

Dr. J. Marion Sims. Clarence K. Weil, Montgomery, Ala. 670

EDITORIALS 672

Technic of Cholecystography
White Bread and Brown
The Suprarenals in Diabetes

LOUISVILLE—WHERE WE MEET 674

Mammoth Cave

BOOK REVIEWS 676

SOUTHERN MEDICAL NEWS 679

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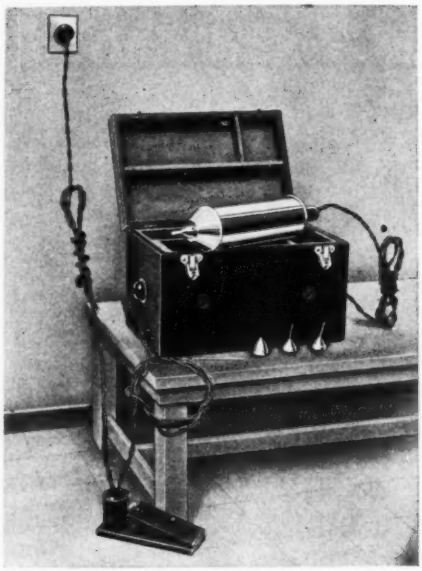

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½ cup hot water	---	---	---	---	---
½ teaspoon salt	---	---	---	---	---
¼ cup vinegar	---	---	---	---	---
1½ cups grated cheese	150	43	54	---	---
½ cup chopped stuffed olives	70	1	19	8	---
½ cup chopped celery	60	1	---	2	---
½ cup chopped green pepper	25	---	---	1	---
½ cup cream, whipped	75	2	30	2	---
Total		51	103	13	1183
One serving		8.5	17	2	197

Soak gelatine in cold water. Bring water and salt to boil and dissolve gelatine in it. Add vinegar and set aside to chill. When nearly set, beat until frothy, fold in cheese, olives, celery, pepper and whipped cream. Turn into molds and chill until firm. Unmold on lettuce leaf and serve.

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	Grams	Prot.	Fat	Carb.	Cal.
1½ tablespoons Knox Sparkling Gelatine	10	9	---	---	---
¼ cup cold water	---	---	---	---	---
1½ cups boiling water	---	---	---	---	---
2 tablespoons lemon juice	20	---	---	2	---
½ teaspoon salt	---	---	---	---	---
1½ cups cooked spinach, chopped	300	6	---	7	---
2 hard cooked eggs	100	13	10.5	---	---
Total		28	10.5	9	242.5
One serving		5	2	1.5	40

Soak gelatine in cold water and dissolve in boiling water. Add lemon juice, salt, strain and chill. When nearly set, stir in chopped spinach, mold and chill until firm. Serve on lettuce hearts or tender chicory leaves and garnish with hard cooked egg, cut lengthwise in sixths and sprinkled with paprika. Serve with mayonnaise.

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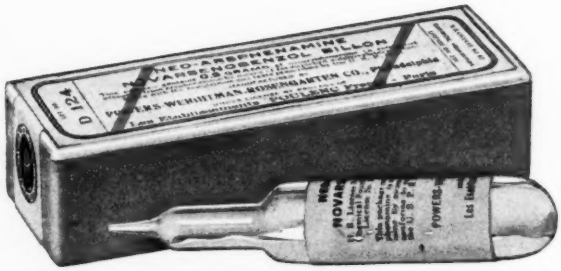

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PELLAGRA This is essentially attributed to deficiency of "P-P" and Vitamine "G" factors of the diet. The remedy of the U. S. Public Health Service, as published by the late Dr. Jos. Goldberger, is Brewers' Yeast-Harris, pasteurized powder —rich in vitamines "P-P" and "G".

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*Yeast Vitamine-B Harris was used successfully with certain of these cases.

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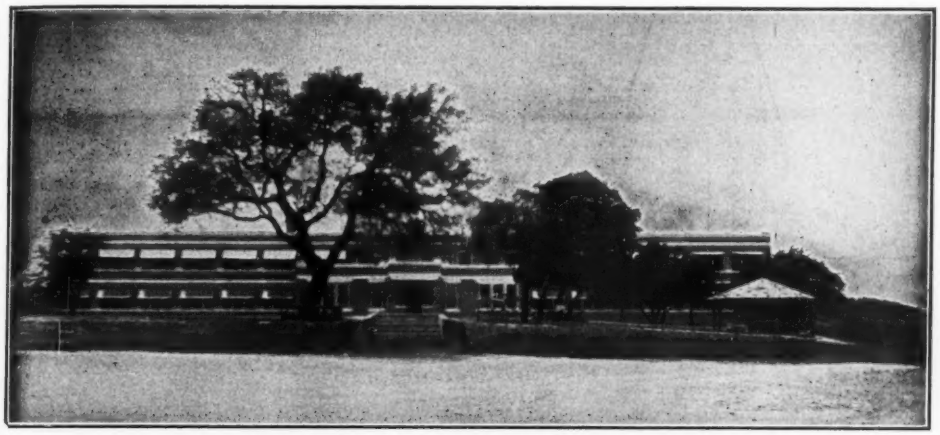

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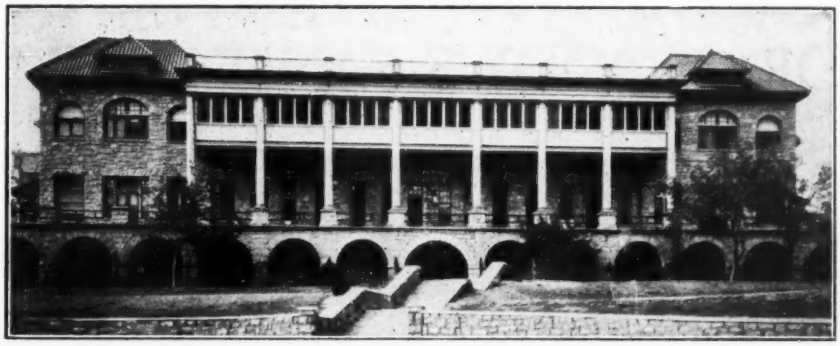

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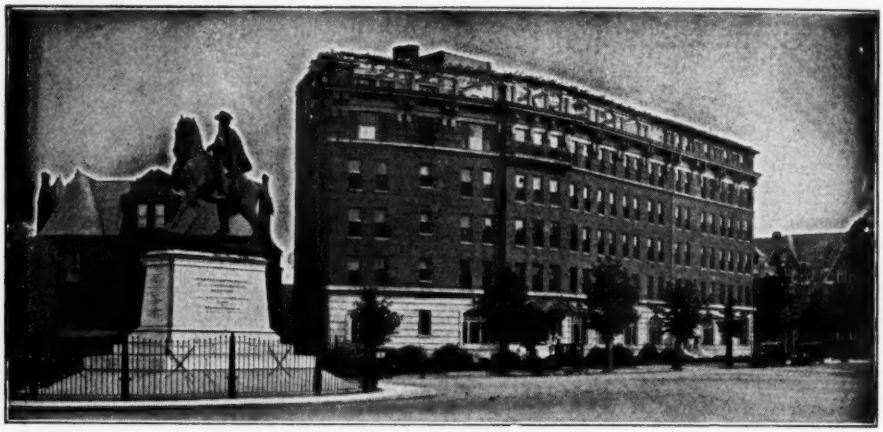

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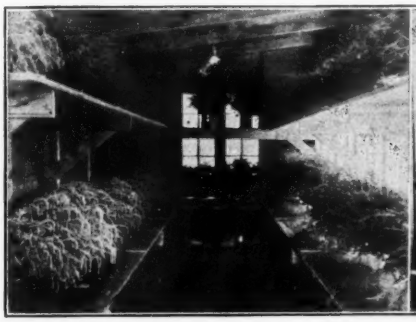

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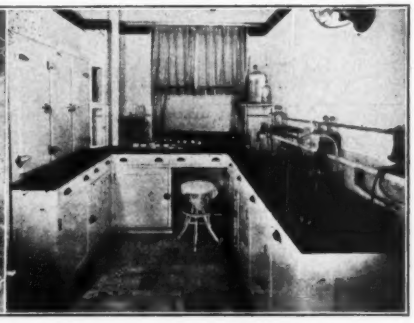

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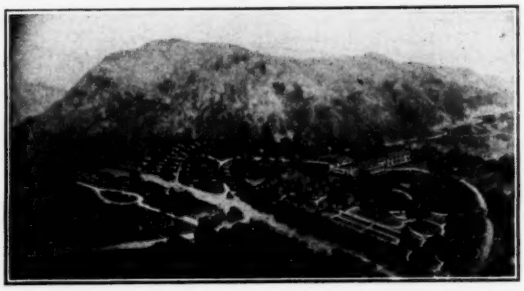

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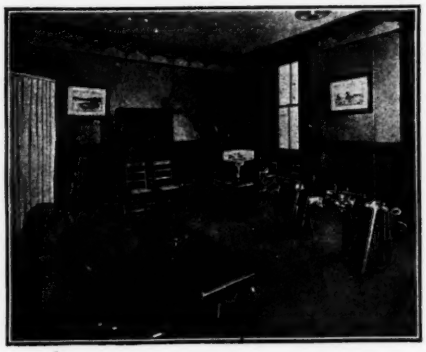

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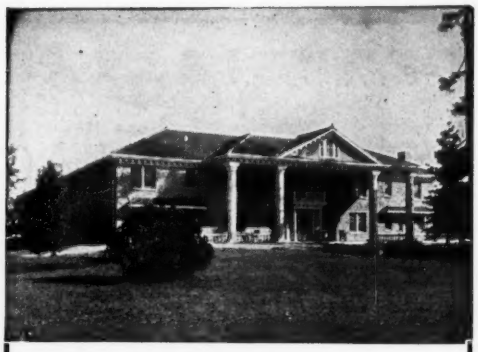

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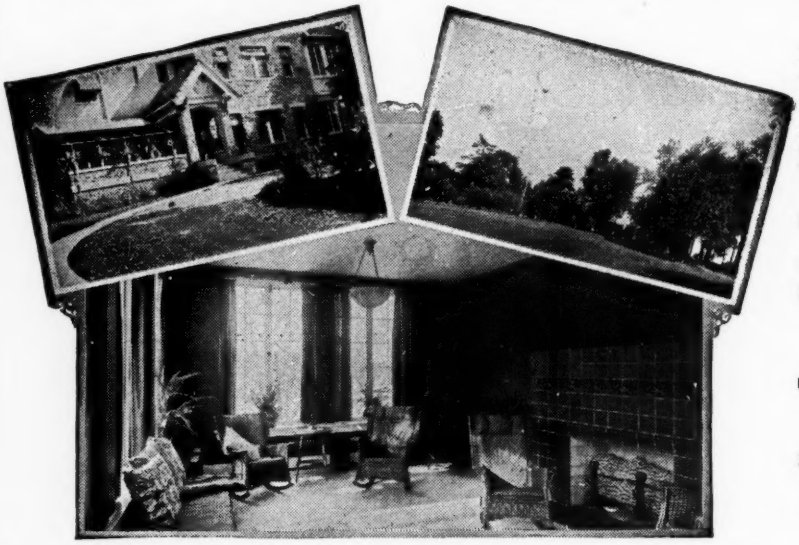

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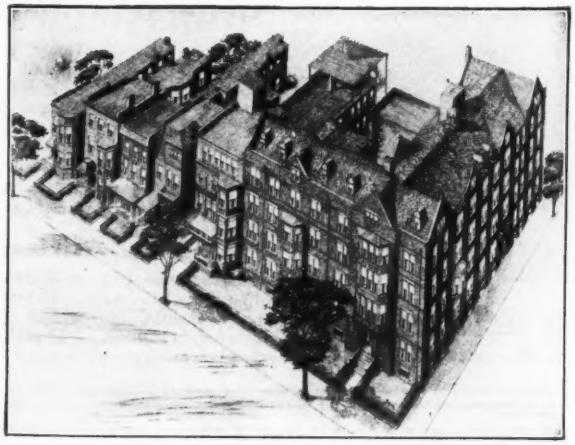

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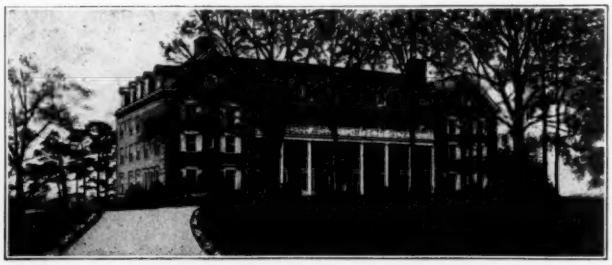

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A CLINICAL AND PATHOLOGICAL STUDY OF POST-MENOPAUSAL BLEEDING*

By RICHARD W. TELINDE, M.D.,
Baltimore, Md.

Vaginal bleeding after the termination of the normal menstrual life of a woman has long been recognized as a symptom calling for serious consideration and experience has taught us that the underlying pathological condition is often some form of malignancy. Indeed, so firmly has this idea been fixed in our minds, that we are prone to forget that, fortunately, not a few benign pelvic conditions may be responsible for this symptom.

From a diagnostic point of view the cases fall into three groups. (1) In the first group the diagnosis is readily made by inspection of the external genitalia, vagina and cervix. As examples may be mentioned senile vaginitis and cervical polyp. (2) In the second group, the diagnosis can be established by microscopic examination of uterine curettings. In this way, adenocarcinoma of the body of the uterus is readily recognized. (3) In the third group, however, careful inspection of the external genitalia, vagina and cervix and microscopic examination of the curettings fail to demonstrate a lesion which would satisfactorily explain the bleeding. In certain of these cases bimanual examination may disclose an ovarian tumor, but in other cases no abnormality can be demonstrated by this method, and indeed one occasionally encounters a case in which no satisfactory explanation of the bleeding can be obtained from the use of all the usual diagnostic procedures.

In order to obtain an accurate idea of the relative frequency of the various lesions responsible for this symptom, I have investigated the histories and pathological records of the cases of post menopausal bleeding treated in the wards of the Johns Hopkins Hospital during the decade from January, 1919, to January, 1929. The results are given in *Table 1.*

From the table it is evident that in our series the most frequent lesion responsible for vaginal bleeding after the menopause was carcinoma of the cervix. This lesion, which occurred in 37.5 per cent of the 179 cases, was more than twice as frequent as adenocarcinoma of the body. It is well known that the latter condition belongs to a much older age than carcinoma of the cervix, but experience shows that even after the menopause the absolute number of cases of carcinoma of the cervix is greater than that of adenocarcinoma of the corpus. In our series there was one case of squamous-cell carcinoma of the body of the uterus, a very rare condition. Thus, in 53.1 per cent of the cases, the bleeding was due to carcinoma of the uterus. If we add to this number the three cases of sarcoma of the endometrium and the two cases of sarcomatous change in myoma, the total percentage of cases in which malignancy of the uterus is responsible for the symptom of bleeding is 55.9. Again, if we add the cases of carcinoma of the ovary, the one case of carcinoma of the endometrium associated with follicular oophoroma, and the one case of carcinoma of the rectum with extension to the vagina (4.1 per cent), the total number of cases in which malignancy in some form is responsible for the bleeding amounts to 60 per cent, leaving 40 per cent in which some benign lesion is responsible.

Primary sarcoma of the endometrium is a rare condition; it was found in only three instances in this series of 179 cases. The ages of the three patients were 66, 52, 49. All had had slight bleeding or a blood-tinged discharge for several months and finally profuse bleeding. Sarcoma had occurred in myoma in two cases and in one case an infected fibroid had given rise to bleeding. It is noteworthy that in not a single case

*Read in Section on Gynecology, Southern Medical Association, Twenty-Third Annual Meeting, Miami, Florida, November 19-22, 1929.

*From the Gynecological Department of the Johns Hopkins University.

TABLE 1

Cases of Bleeding After the Menopause

		No.	Per Cent	No.	Per Cent
Carcinoma of cervix	squamous-cell	45	25.1	67	37.5
	adenocarcinoma	5	2.9		
	type undetermined		9.5		
Carcinoma of body of uterus	adenocarcinoma	27	15		
	squamous-cell carcinoma	1	0.6		
Sarcoma of endometrium		3	1.7		
Sarcomatous change in myoma		2	1.1		
Infected myoma		1	0.6		
Prolapse with ulceration of cervix		20	11.2		
Ulcer of cervix (etiology undetermined)		1	0.6		
Cervical polyp		12	6.7		
Endometrial polyp		8	4.5		
Chronic endometritis		6	3.4		
Hyperplasia of endometrium		3	1.7		
Chronic senile vaginitis		4	2.2		
Ulceration of vagina from pessary		2	1.1		
Chronic ulcerative vulvitis (etiology undetermined)		1	0.6		
Granuloma inguinale		1	0.6		
Urethral caruncle		1	0.6		
Urethral prolapse		1	0.6		
Carcinoma of rectum with extension to vagina		1	0.6		
Normal endometrium; no gross pathology; cause of bleeding not determined		5	2.8		
Ovarian tumors	Fibroma	1	0.6		
	Cystadenoma	2	1.1		
	Adenocarcinoma	1	0.6		
	Adenocarcinoma, extension to uterus	5	2.9	12	6.7
	Granulosal-cell tumor	2	1.1		
	Follicular oophoroma with carcinoma of endometrium	1	.6		

TOTAL 179 Cases

had an uncomplicated fibroid given rise to bleeding after the menopause. Hence, the finding of a fibroid in a bleeding woman after the cessation of her periods does not satisfactorily explain the symptom, for further investigation will often disclose an additional and not improbably a more serious lesion, due to some change in the fibroid itself or to an independent accompanying condition.

Prolapse with ulceration of the cervix was responsible for bleeding in 11.2 per cent. The character of the bleeding in these cases is usually a blood-streaked discharge rather than a frank hemorrhage. Examination of the protruded cervix will disclose an ulcer as the source of the hemorrhage, but in case one cannot satisfy himself that this is the entire source of the bleeding, further investigation becomes imperative. In one of our cases of ulcer of the non-prolapsed cervix, the microscopic picture gave us absolutely no clue to its etiology. Bleeding from cervical polyps may be moderately profuse, but the usual history is of a slight show, especially after intercourse, defecation or any straining. Benign endometrial polyps may occur after the cessation of the menses and in this series were responsible for the bleeding in 4.5 per cent. The bleeding from this condition is also usually scanty, but one patient in this series, a woman of 85, was bleeding profusely from a large benign polyp high up in the uterine cavity. Chronic endometritis was responsible for bleeding in 3.4 per cent (six cases). In five of these six cases of endometritis, pyometrium was found at operation. In the sixth case no mention was made of it. The etiology of the pyometria in these five cases was a benign stenosis of the cervical canal due to senile contracture of the cervical tissues. The usual history in these cases was of a blood-tinged, purulent discharge. The microscopic picture of the endometrium shows a senile endometrium, densely infiltrated with inflammatory cells, chiefly of the round-cell variety, often with a marked metaplasia of the surface and glandular epithelium. In rare cases the

Fig. 1
Chronic endometritis. Note the round cell infiltration and the complete change of the surface epithelium to the stratified squamous type.

columnar epithelium is completely changed to the stratified type (see *Fig. 1)*.

The three cases (1.7 per cent) of hyperplasia of the endometrium are of special interest, for, according to our usual conception of this condition, its occurrence is confined to the years of active menstrual life. This age incidence supports the present day conception of the condition which regards the underlying cause as disturbed ovarian function. Both Meyer and Schroder independently came to the conclusion that the cause of hyperplasia of the endometrium is an absence of the influence exerted by the corpus luteum and a persistence of the follicular influence. They base their conclusions on the demonstration, in a considerable amount of their pathological material, of a complete absence of corpora lutea but an abundance of cystic follicles. Assuming this to be correct, its occurrence would presuppose the presence of follicles in the ovary. It is noteworthy that the three cases in our series were found in two patients at the age of fifty and in one at the age of fifty-one, the three cases occurring two years, fourteen months, and six months, respectively, after the last menstruation. In other words, the cases all occurred shortly after the cessation of the menses and are probably indicative of a partial persistence of ovarian function. Just as some women may have what is apparently a menstrual period several months or a year after the last period, due to development and retrogression of a final corpus luteum, so the persistence of a last follicle might bring about hyperplasia of the endometrium with prolonged bleeding several months after the last period.

Chronic senile vaginitis gave rise to bleeding in four cases in this series. The actual incidence of this condition is probably much greater, as these statistics are taken from hospitalized patients and the majority of patients with this condition are not admitted to the hospital. These patients usually have a blood-tinged discharge rather than frank bleeding. The importance of this condition, from a diagnostic point of view, lies in making certain that the bleeding is entirely explained by the vaginitis and that there is no other more serious lesion higher up in the generative tract. If there is any doubt in the examiner's mind as to whether the vaginitis is the sole cause of the bleeding, a curettage is indicated. There were two cases of ulceration caused by a pessary. This figure also is probably not a true indication of the frequency of this condition, for these women usually are not hospitalized. Chronic ulcerative vulvitis (etiology undetermined), granuloma inguinale, urethral caruncle and urethral prolapse were each responsible for genital bleeding in one instance.

In five instances in which there was a history of bleeding after the menopause nothing could be made out on pelvic examination, and the curettings showed normal, senile endometrium, leaving us unenlightened as to the explanation of this symptom. (For a possible explanation see discussion.)

In this series of 179 cases there were twelve ovarian tumors associated with post-menopausal bleeding. It may be noted that these tumors were of various histological types: fibroma, cystadenoma, adenocarcinoma, granulosal-cell tumor and follicular oophoroma. These twelve cases occurred in a series of 41 ovarian tumors which occurred after the menopause. In five of the cases the bleeding is readily explained by an extension of the carcinoma to the uterus. In the other seven this was not the case and so simple an explanation of the bleeding could not be accepted. That bleeding does occur with various types of ovarian tumors is borne out by a survey of the literature. Robert Meyer reported a round-cell sarcoma and a fibroma of the ovary associated with post-menopausal bleeding. Moulonguet-Doleris found bleeding in 19 of 74 cases of ovarian tumors of various types after the

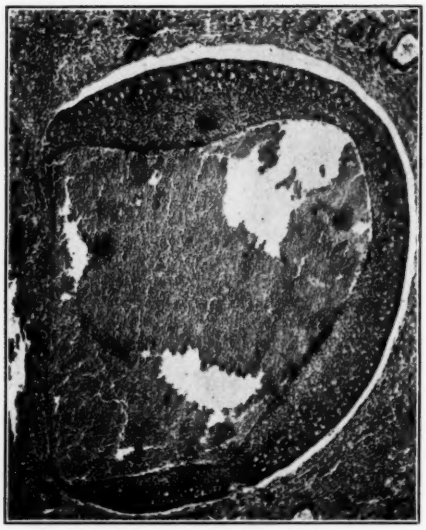

Fig. 2
Follicle-like structure from granulosal cell tumor.

menopause. Schiffmann, in three publications, reports nine cases of post-menopausal bleeding associated with carcinoma of the ovary. Schiffmann's cases are particularly instructive, for they have been, for the most part, followed over a sufficient period of time, permitting the author to arrive at definite conclusions.

In one of his cases a sixty-six-year-old woman, who had had her last menstrual period ten years before, bled slightly for one day only. A curettement showed an atrophic endometrium. A vaginal hysterectomy was performed. No malignancy of the uterus was found. Two years later she returned for bleeding and pain. On examination a tumor was found in the left ovarian region. A sinus from the tumor to the vagina was curetted and alveolar carcinoma was demonstrable. The patient died under x-ray treatment. Schiffman concluded that this tumor was present when the patient had presented herself two years before for bleeding, but that it was so small that no enlargement of the ovary could be made out by bimanual palpation at that time.

A second case reported by Schiffmann was of a seventy-year-old woman whose last menstruation had occurred eighteen years before. She complained of very scanty vaginal bleeding which had lasted for several days. Pelvic examination revealed nothing striking. A curettement showed no evidence of malignancy. Recalling the previous case he performed a laparotomy and found a very slight enlargement of the right ovary which microscopically showed carcinoma.

A third case reported by Schiffmann is that of a fifty-three-year-old woman who began to bleed three years after her last menstrual period. The bimanual examination was negative. She consulted another gynecologist who curetted her and decided from the curettings that nothing more was necessary. Five weeks later she again presented herself to Schiffmann with a large tumor in the cul-de-sac. Laparotomy was performed and carcinoma of the ovary found. The patient died three weeks later.

These three cases, and others reported in the literature, indicate to Schiffmann that post-menopausal bleeding may be an early symptom of carcinoma of the ovary, and that when the symptom cannot satisfactorily be attributed to the uterus, careful watching for a palpable ovarian tumor is necessary and a laparotomy should be done as soon as the slightest ovarian enlargement is noted.

Of particular interest are the three cases in our series of the follicular type of ovarian tumor. Two of these are of the granulosal-cell type of tumor *(Fig. 2)*; the third is of the type described by Brenner as follicular oophoroma *(Fig. 3)*. Both are composed of structures which, at least superficially, resemble the Graafian follicles. In the granulosal-cell tumors the foreign cells closely resemble the granulosal cells of the normal follicle. In the follicular oophoroma the tumor cells are larger and polygonal or spindle in shape. In neither of these two types are cells resembling ova found in the follicle-like structure. Both types of tumors have a dense fibrous stroma which has been invaded by the tumor cells, but both have a well developed fibrous capsule. Although the tumors are very cellular the cells for the most part are quite uniform in size and staining qualities. No mitotic figures are seen in either type of tumor.

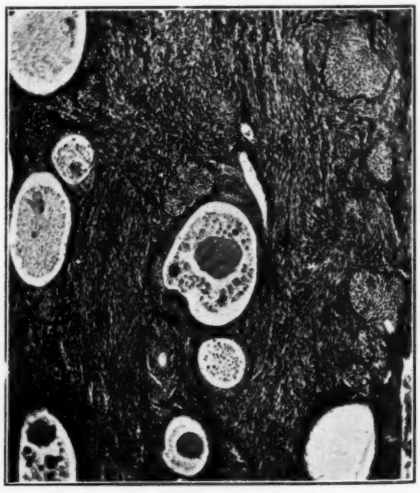

Fig. 3
Follicular oophoroma. Note the dense fibrous stroma, the solid islands of epithelium and the folliculle-like structures.

The relation of these tumors to post menopausal bleeding is of special interest. The three cases in the series were all associated with rather profuse recurrent attacks of bleeding which the patients described as being at least as profuse as their normal menstruation. In one of our cases of granulosal-cell tumor, the patient, who had ceased menstruation twenty-two years before, began to bleed for a few days, approximately once a month, over a period of six months. In the other case of granulosal-cell tumor the patient began to bleed four years after the cessation of her normal menstrual life and bled at irregular intervals (one to three weeks) for four years. In the case of follicular oophoroma, the patient, aged 63, stated that she had never ceased menstruating. On close questioning, however, it was found that eight years previously she had failed to menstruate for a period of eight months. This cessation probably represented her menopause. She then began having recurrences of vaginal bleeding at irregular intervals of several weeks. Three months before operation she had bled continuously and profusely, but this was undoubtedly due to an accompanying carcinoma of the endometrium. In the one case of granulosal-cell carcinoma, in which the uterus was available for study, the viscus was as large as a uterus of a woman in active menstrual life and the endometrium showed marked hyperplasia. Likewise, in the follicular oophoroma the uterus was large, in spite of the patient's age (sixty-three years), and between the areas of carcinoma of the endometrium were large benign dilated glands suggestive of hyperplasia. This finding of hyperplasia of the endometrium in granulosal tumors has been frequently reported in the literature and would apparently indicate some hormonal influence of this specific type of tumor.

As to the malignancy of these tumors there is some difference of opinion. Brenner regarded his case of follicular oophoroma as benign. Our patient is well one year after operation. We are inclined to agree with Brenner, for in our patient, who gave a history of probably eight years' duration, the tumor was well encapsulated and perfectly free in the abdominal cavity. Although the tumor cells presented a picture suggesting invasion of the fibrous stroma of the tumor, they were remarkably uniform in size, shape, and staining qualities. The granulosal-cell tumors have been called carcinomatous by certain authors, we have found only three cases in the literature in which recurrences are reported, the remaining cases being living and well one to eleven years after operation. Our patients have remained well for two and three years after operation, notwithstanding the fact that in one there was a history of four years' duration and the tumor was a huge structure weighing 20 pounds. In spite of this size it was perfectly encapsulated and non-adherent. Hence, for the present we are content to label them granulosal-cell tumors, remembering that a certain degree of potential malignancy exists.

From this clinical and pathological study, we have come to the following conclusions:

(1) In our series of 179 cases of post-menopausal bleeding, malignancy was found somewhere in the generative organs in 60 per cent and relatively benign conditions in 40 per cent.

(2) The most common site of the malignancy is the uterus (55.9 per cent).

(3) Certain benign and malignant causes of genital bleeding after the menopause can be diagnosed by inspection of the vagina and cervix or curettage of the uterus, but in a small group of cases these procedures do not satisfactorily explain the symptom.

(4) When speculum examination of the vagina or curettage of the uterus fails to explain the cause of bleeding satisfactorily, the question of neoplasm of the ovary should be borne in mind; and even though no evidence of ovarian neoplasm can be discovered on palpation at the time of the curettage, inasmuch as the presence of a very small tumor is quite possible, the patient should be subjected to re-examination frequently and as soon as any increase in size of an ovary is made out, she should be subjected to a laparotomy, provided her age or other physical condition does not present any serious contra-indication.

(5) If curettage reveals hyperplasia of the endometrium several years after the menopause, the presence of follicular oophoroma or a granulosal-cell tumor of the ovary is very probable, and the patient should be kept under observation and subjected to laparotomy as soon as palpation shows any enlargement of the ovary.

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DISCUSSION *(Abstract)*

Dr. Erasmus H. Kloman, Baltimore, Md.—The five undiagnosed cases may in part, at least, fall into a group which seems unmentioned in the paper, namely: those cases of arteriosclerosis, which I see rather frequently and I believe should be classed as compensatory bleeders trying to relieve their high blood pressure, the cases the laboratory most frequently reports as adenomyoma.

One wonders whether granulosal-cell tumors of the ovaries always cause bleeding. Do ovarian tumors in general cause uterine bleeding? I am convinced that we do get occasional bleeding from the simple cysts of the ovary, but not all ovarian tumors cause bleeding nor do they cause any symptoms unless they be from pressure or twist. Dr. TeLinde removed the uterus as well as the tumor from *cases 1* and *3*, but not from *2*. It would be interesting to know whether all bleeding ceased in *case 2*. It would be of interest to compare the results of a group of cases where the tumor alone was removed with another where the tumor and uterus were removed.

One of the important points brought out by this paper is the necessity for the correct conception of the physiology of menstruation and ovulation, and until we have the proper understanding of this phenomenon, we shall not understand this and most other types of uterine bleeding, nor shall we be able to treat them as intelligently as we should like.

Dr. Willard R. Cooke, Galveston, Texas.—Many cases of post menopausal bleeding are carelessly diagnosed as "polyp" of the endometrium. This diagnosis is rarely correct. Incidentally, the term polyp can very well be dispensed with, since it is an expression loosely used to indicate all sorts of unrelated processes, the treatment of which is as variable as are the pathological conditions.

In a case of this sort, when the lesion is visible, it is easy to determine just what one is dealing with by histologic examination. Unfortunately, many practitioners will encounter lesions of doubtful appearance and cauterize them or treat them in some other way without histologic examination, until the carcinoma becomes quite advanced. I consider that all cases of post menopausal bleeding should be subjected to histologic examination, either of sections from suspicious looking areas, or of scrapings from the endometrium. In this connection, it must be borne in mind that histologic diagnosis of endometrial lesions is not always dependable. We have had several cases in which biopsy specimens of the endometrium were reported as simple adenomata. The clinical picture in these cases, however, was so suspicious that they were treated as carcinoma; and on examination of the uterus after removal it was found that whereas the periphery of these growths was wholly benign in microscopic appearance, there was indubitable cancer in and about the base of the growth.

We have had three cases of post menopausal bleeding from ovarian conditions. The first two were simply bleeding from invasion of the uterus by massive advanced carcinoma primary in the ovary. The last case occurred rather recently, and we hoped that we would find a folliculoma. This patient had started a fairly regular cyclic bleeding some time after menopause. The endometrial scrapings were reported by our pathologist as hypertrophic endometritis. On examination we were, ourselves, unable to classify the lesion. On removal of the ovary we found a very thoroughly encapsulated early carcinoma simplex.

Dr. TeLinde (closing).—When I started this investigation, arteriosclerosis was one of the things that I had particularly in mind. It is referred to in the literature quite frequently. But it has always impressed me that the pathological reports of it were not particularly sound. In the five cases of bleeding that I did not explain on a satisfactory pathological basis the average age was fifty-one years. The average systolic and diastolic blood pressure was 139/85, so certainly there was no hypertension in these cases. Although I cannot deny the existence of uterine hemorrhage in hypertensive cardiovascular disease, I do not know of any sound pathological evidence which substantiates it.

EGGS AND LARVAE OF COMMON HOUSE FLY FOUND IN HUMAN INTESTINE*

PRELIMINARY REPORT

By MARVIN SMITH, M.D.,
Miami, Fla.

During the past two years or more I have frequently seen egg-like bodies appear on glass slides which we used for "set-ups" in the examination of feces.

Our laboratory routine used in searching for intestinal parasite eggs is to dissolve a portion of feces about the size of a lima bean in one ounce of saturated sodium chloride solution; we next fill a one-ounce medicine glass with this emulsion and place over it a wide glass slide which just touches the watery emulsion and allow this to stand for twelve to twenty-four hours, at the end of which time the slide is taken off and examined with the 2/3 objective for eggs.

The egg-like bodies referred to are ovoid in shape with a more or less crumpled shell membrane, and are about 30 m'crons in diameter or approximately that of the short axis of a hook worm egg.

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*Read in Section on Gastro-Enterology, Southern Medical Association, Twenty-Third Annual Meeting, Miami, Florida, November 19-22, 1929.

Eggs of Aedes aegypti as seen by electrolyte
Eggs of Aedes aegypti as seen by electrolyte
Eggs of Aedes aegypti greatly magnified
Eggs, hatching cut, five days after planting
Larvae, three days old
Larvae, six days old
Larvae 10 days old, full grown
Pupation stage, twelve days after eggs hatched
Adult Aedes aegypti, (female fly), 16 days after eggs hatched

Many slides showing these bodies were seen before any effort was made to determine what they were. After subjecting the specimen of feces to the above described technic, it is not uncommon to see on a single slide from three to fifty eggs.

We plated some of the feces containing the eggs on both Ayers-Rupp and Torrey's media. At the end of forty-eight hours we could find nothing that resembled larvae, so after conducting similar tests on numerous other specimens with eggs and finding no larvae we decided to remove the plates from the incubator and place them in an enclosure carefully screened. There they remained for thirty-six hours more, making one hundred and twenty hours in all, at the end of which time the larvae hatched out in large numbers. Some of these larvae were then transferred to a well-screened container in which we placed a mass of decaying and fermenting bananas and apples, the mixture being slightly moistened with a little water. In another vessel similarly arranged, was placed finely chopped fresh beef.

On both classes of foods, the larvae grew and thrived for ten or twelve days, thus demonstrating to some extent their omnivorous tendencies. The larvae grown on beef mixture were most thrifty and dissection showed that their digestive tract contained numerous red blood cells which they undoubtedly consumed for their food. A little water was added to the decaying meat and fruit from time to time to maintain a decidedly wet, mushy mixture. Close observation showed that none of the larvae changed to the puparial state as long as the soupy medium was maintained. Finally a few small pebbles were put into the mixtures so that the larvae could crawl upon them, and there the integument of their bodies gradually dried and shrank and in three days they entered the puparial state.

The pupae were then placed in a tightly screened container which had slightly moistened sand in it and in ten days the adult flies developed. To my surprise and astonishment, they proved to be the *musca domestica* or common house fly showing the typical maroon-colored heads and longitudinally striped thorax and abdomen, dorsally. The larvae, we found, would live in either acid or alkaline feces.

During the months of August and September, 1928, six male and eight female patients were studied, care being exercised to avoid the possible contamination of the specimens by exposure to flies after defecation. Eggs were found in all of these in considerable numbers and during the same months plantings on sterile media from seven other male and seven female patients likewise hatched out larvae.

Upon finding the eggs in feces from many individuals, I concluded to try giving various anthelmintics, thinking that some of the patients might not only possess the eggs, but the larvae also, but nothing brought any larvae out except the oil of chenopodium. I administered it in five drop doses and gave one capsule every hour until three were given, then followed with a saline cathartic, which treatment brought the larvae out dead. Of fifteen egg-positive cases so treated, only three were found with the larvae in the colon. In one of the cases treated, about 200 well grown larvae were expelled in one day, and in about ten days another treatment brought out about 300 more. In a second case about 75 were recovered and in the third positive case about 35 were found.

In this preliminary report I wish to make grateful acknowledgment to Dr. Frank H. Harms of Orlando, Florida, who furnished numerous specimens from a very interesting case for study and investigation.

CONCLUSIONS

(1) The eggs and larvae of the *musca domestica* or common house fly can and do exist in the human intestine without regard to the reaction of the feces.

(2) The eggs are ingested on foods either cooked or raw on which flies are allowed to alight for even an instant.

(3) Many eggs remain in the human digestive tract long enough to hatch and develop into full-sized larvae.

(4) These larvae probably remain more or less indefinitely, hidden in the folds of the mucous membrane of the colon.

(5) The larvae form appears to require a rather dry place in the open atmosphere in order to dry and pass into the puparial stage.

(6) Every human being in the world who eats food upon which flies have been permitted to alight is susceptible. The eggs and larvae are very much more prevalent during the active laying season of the fly, from April to September.

(7) Oil of chenopodium, followed by a saline cathartic, is fairly effective in removing them.

(8) The maggots are omnivorous and have been found with the enteron filled with red blood cells.

(9) Every patient from which the larvae have been removed suffered from marked secondary anemia.

(10) A complete report cannot be made at this time as to how much or in what special manner the human body is influenced by the presence of these larvae or eggs.

REMARKS

A point of some interest is that a more or less dry environment is essential to the change from the larvae to the pupae stage since this is accomplished by a drying and shrinking process.

In Stitt's work on "Parasitology", *Auchmeromyia luteola*, found in Africa, is cited as the only known species of blood sucking larvae.

In this preliminary report I hope not to make any unwarranted statements, but I have undertaken to bring proof, at least to my satisfaction, that larvae of the common house fly can and do exist in the human intestine and that these larvae, while not possessing piercing organs, adapt themselves to their environment and with their powerful rudimentary sucking proboscis draw blood for food from the soft mucous membrane of the colon when the opportunity presents itself.

It is my feeling that we should give more attention to intestinal myiosis than we are accustomed to give, and it is my belief that the more we study the habits of the common house fly, the greater enemy of the human family we shall find him to be.

405-6 Huntington Building

DISCUSSION *(Abstract)*

Dr. J. E. Knighton, Shreveport, La.—Several years ago a patient brought a specimen of larva, which proved to be that of the common house fly, to my office which she was sure that she had passed with a bowel movement after having taken an enema. It occurred to me that the patient was probably mistaken and that the feces had been contaminated after evacuation.

Many years ago when I was practising in a small community that was not provided with a sewer system, I frequently observed specimens of feces that were simply alive with very active larvae of a fly which I was not able to identify. This fly was somewhat larger than the house fly and was gray in color. The ova of this fly, when deposited on fresh feces, hatch out immediately and become quite active. Within a remarkably short time they develop into full size larvae.

It has been worth while for Dr. Smith to bring this matter to our attention on account of its possible bearing on the sanitation of our food supplies.

Dr. W. S. Anderson, Memphis, Tenn.—In the Section on Railway Surgery yesterday rather an interesting topic was brought up along this line. Those of us who do surgery, particularly in the negro wards of the hospital, have been unfortunate enough at times to have our wounds contaminated by the house fly so that the maggot will develop. Sometime ago a paper was read reporting contamination of an old fistulous sinus, and also, of a case of chronic osteomyelitis by maggots. The maggots were removed and the wound healed. Later maggots were placed purposely in old suppurating wounds, and it was found that they destroyed only the infected tissues. On examination of the maggots themselves no bacteria were found. I am told that now at Johns Hopkins Hospital they are beginning to cultivate these maggots and to apply them intentionally in certain types of cases.

Dr. W. E. Vest, Huntington, W. Va.—I am told that Dr. Baer at Hopkins has been making investigations with the maggots referred to by Dr. Anderson, inviting them into the old suppurating wounds.

Dr. Smith (closing).—It is reassuring to learn that other observers have at least had their curiosity aroused by finding maggots in human feces. Every now and then reliable patients have reported to me that they have seen full-grown larvae in their own defecation.

The fly referred to by Dr. Knighton is larger than the house fly and gives birth to maggots while the *musca domestica* lays eggs from which the larvae hatch.

Mosquitoes harassed civilized man for untold centuries before it was definitely proved that they played a role in the causation and dissemination of disease. Since we know that the house flies are the most common of all insects among all peoples of the earth, and since we are familiar with their loathsome habits, we might do well to scrutinize them a little more carefully. We know that their eggs and larvae can and do live in the human intestine and that the larvae are blood suckers. I hope to make additional reports later as my study along this line proceeds.

THE CYSTOSCOPIC APPEARANCE OF VARIOUS TYPES OF CYSTOCELES*

By Rex E. Van Duzen, M.D.,
Dallas, Tex.

I have been consulted by women frequently because of recurrent attacks of so-called "cystitis" or "trigonitis," who give the history of having had one of the various types of operations designed for repair of cystocele. They say: "The doctor says the operation looks perfect, but I am not relieved of the bladder irritation."

About three years ago I saw a woman who gave the history of having had a perineorrhaphy, in whom the symptoms of constant desire to urinate and pressure on bladder and rectum were exaggerated following operation. Examination showed an over correction of the perineum which necessitated depression of it before the urethra could be seen, and which made vaginal examination impossible without severe pain to the patient. Cystoscopic examination revealed a large cystocele just posterior to the internal urethral sphincter. Both ureteral orifices were situated on the posterior wall of the bladder with a well developed interureteral muscle (Mercier's bar) but the portion of the trigone extending down into the deep urethra was not demonstrable. I then withdrew my cystoscope to view the internal sphincter, hoping to find some cause for the delay in emptying her bladder. The patient was instructed to empty her bladder and it was noted that there was a delay of several seconds between contraction of the interureteric portion of the trigone and the depression of the internal sphincter. It was also noted that the ureteral orifices did not draw downward toward the internal sphincter, but on the contrary retracted upward on the posterior wall of the bladder with accentuation of the interureteric muscle.

After a rather complete review of the literature, I found that Young and Wesson most thoroughly discuss the function of the trigone muscle in males, but I have been unable to find any references save the briefest remarks concerning its function in females. Young and Wesson describe the male trigone as follows:

"The trigone is a triangular area lying in the base of the bladder, having the two ureters and the vesical orifice at its angles. In the trigonal area are the two muscle layers of the bladder, the external longitudinal and the internal circular, and superimposed upon these is the submucous or trigonal muscle, which is an extension of the longitudinal muscle layer of the ureters and their sheaths. It consists of fibers extending fanwise from the ureteral orifices, some fibers passing medially going to make up Mercier's bar, others interlacing with corresponding fibers from the opposite side in the middle of the trigone, and still others passing down to the urethra and making up Bell's muscles. The Bell's muscles of the two sides converge at the vesical orifice, causing the trigonal muscle at this point to be denser and thicker. Where the thickened layer passes over the edge of the vesical orifice it makes up the principle portion of the uvula of Lieutaud.

During the last three years I have made the examination of the action of the trigone and internal sphincter a routine part of every cystoscopic examination, in both males and females. Briefly, this examination consists of withdrawing the Brown-Buerger cystoscope until the internal sphincter fills the lower third of the field. By lowering the objective end of the cystoscope the base of the bladder is brought into view as are usually both ureteral orifices. One notes:

(1) The promptness with which the internal sphincter depresses when the patient tries to void.

(2) Whether Bell's muscle becomes more prominent or less.

(3) Whether ureteral orifices draw inward and toward the internal sphincter or backward and inward.

(4) Whether in female patients in whom a cystocele is present it is anterior or posterior to Mercier's bar.

For ease in discussion I have divided these cases into:

(1) Normal.

(2) Cases with a cystocele, in which there is a partial or complete division of Bell's muscle and retraction of muscle upward.

(3) Cases with a cystocele in which there is a partial or complete division of Bell's muscle and retraction downward.

(4) Cases with a cystocele posterior to the trigone.

(5) Cases with general relaxation of the entire base of the bladder.

(6) Cases in which the cystocele involves the internal sphincter and posterior urethra.

(1) *Normal.*—Young and Wesson state:

"The opening of the internal sphincter during micturition was formerly considered as an inhibitory action. We now believe that the contraction of the powerful trigonal muscle passing in the form of a bow through the weaker arcuate muscles at the vesical orifice helps mechanically to open the sphincter."

I try to divide the act of voiding in, first the contraction of the trigonal muscle and lowering of the internal sphincter; second, the contraction of the muscles of the bladder wall; and lastly, the contraction of the abdominal muscles. When the trigonal muscle is destroyed the opening of the sphincter is delayed. This was beautifully

*Read in Section on Urology, Southern Medical Association, Twenty-Third Annual Meeting, Miami, Florida, November 19-22, 1929.

Fig. 1 (top). Normal. Note position of urethral sphincter.

Fig. 2 (bottom). Normal when attempting to void. Note depression of urethral sphincter and prominence of trigone.

shown in a case in which I unwittingly paralyzed the trigone by cutting the presacral nerve, the hypogastric sympathetic nerve, which when stimulated causes contractions of the trigone muscle. This operation was performed in attempting to cure an obstinate case of masturbation. Since operation there is a delay in starting the stream and it is necessary for the patient to strain in order to empty bladder. Overstretching of the urethra relieves this difficulty for three or four weeks, but not permanently. I believe the necessity of increasing the inter-vesical and inter-abdominal pressure in starting the act of urination rather than depression of the sphincter by the trigone muscle account for many recurring cystoceles.

(2) *Patients with a cystocele in whom there is a partial or complete division of Bell's muscle and retraction of the muscle upward.*

Case Report.—Mrs. D., age 56, para four, consulted

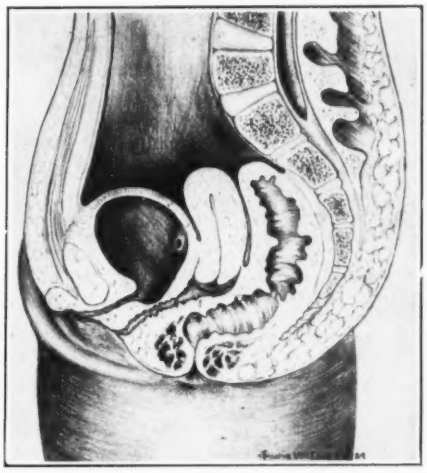

Fig. 3. Normal. Sagittal section.

Fig. 4 (top). Division of Bell's muscle with retraction upward.

Fig. 5 (bottom). Same case on straining to void.

me for painful urination, blood in the urine and a constant sense of pressure in the bladder. Cystoscopic examination showed a moderate sized cystocele between the internal sphincter and Mercier's bar. Bell's muscles were divided and retracted upward (*Fig. 4*) and on attempting to void there was a marked delay in depression of the sphincter. Furthermore, the retracted muscle drew up further into a small tubercle (*Fig. 5*). The patient was operated upon by plicating the vesical fascia, the surgeon carrying this well down under the deep urethra. This case had lost the action of the trigone muscle and the sphincter was opened by the pull from the base of the bladder, resulting in the delay. Before the operation she had residual urine with incrustations at the base of the bladder. The operation has relieved her of the residual urine, but has not altered the opening of the sphincter. However, this condition was explained to her before the operation, and she accepted it.

(3) *Patient with a cystocele in whom there is a partial or complete division of Bell's muscle and retraction of the muscle downward.*

Case Report.—Mrs. C. B. G., age 58, para three, came complaining of pain in the back radiating to the bladder, and difficulty in passing urine. The urine would start and then stop suddenly, necessitating a change in position, and the patient had to relax before urination started again. This was repeated several times during the act of urination. Cystoscopic examination showed a normal urethra. The bladder wall showed moderate trabeculation. The trigone was very prominent and extended into a large wart-like growth which could be felt through the vagina. This bled freely, but was covered by normal epithelium. When the patient attempted to empty the bladder this was drawn up into the field, nearly filling the field of vision. It was removed by fulguration. One year later the tubercle could not be seen, and starting and stopping of the stream had ceased.

I have seen three such cases. It was suggested to me that I was dealing with a granuloma or a hypertrophied uvula and not the retracted portion of Bell's muscle, but in one case seen six months after her only delivery, contractions of muscle fibers could be plainly seen in the retracted stump. Again especially in men with a prominent uvula, we see the tubercle flatten out when the muscle contracts rather than become more prominent as in this case.

Two cases were completely relieved by fulguration of the retracted muscle. The third still has dribbling of urine at times. The long stretching of the muscle may account for this partial incontinence.

(4) *Cases with a cystocele posterior to the trigone.*

Case Report.—Mrs. S., age 26, para two, was under treatment for pyelitis when she became pregnant. She was delivered of twin girls and fifteen months later was delivered of a single girl baby. Six months after the second delivery she was cystoscoped, at which time the

Fig 6 (top). Division of Bell's muscle with retraction downward. (Picture is upside down.)

Fig. 7. (bottom). Same case on straining to void.

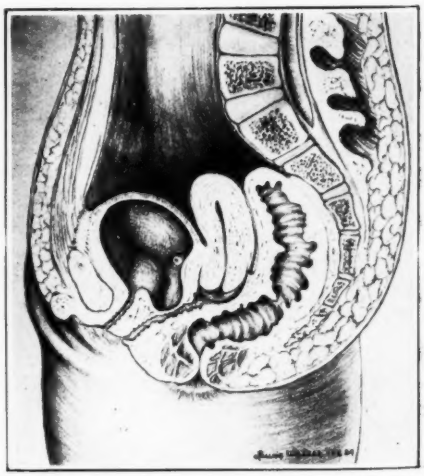

Fig. 8. Sagittal section of cystocele anterior to Mercier's bar.

Fig. 9. Case showing cystocele posterior to trigone.

Fig. 10. Sagittal section of same.

trigone was found to be very prominent and immediately behind there was a large cystocele which stopped abruptly at Mercier's bar. This patient has no delay in emptying the bladder, but was subject to recurrent attacks of cystitis. This is the type of patient who should obtain complete relief by repair of the cystocele.

(5) *General Relaxation of the Entire Base of the Bladder.*—This is a very common condition in which we see pseudodiverticulae behind the ureteral orifices and the entire base of the bladder in folds except when the bladder is in extreme distension. The superior wall of the vagina anteriorly may look normal, but in the posterior vagina near the cervix we find marked relaxation, often making it difficult to show the cervix. These cases may be symptomless, but usually give the history of repeated attacks of cystitis and often of residual urine.

(6) *Cases in which the cystocele involves the internal sphincter and the posterior urethra.*

Case Report.—Mrs. S., age 34, para one, came under observation because of frequent and painful urination. This is especially worse when she is on her feet. The patient states that since the birth of her child she has had to relax rather than to strain in order to void. It is also necessary to lean forward almost to a prone position because the stream is directed upward toward the abdomen.

Examination showed a marked cystocele which involved the posterior urethra. Cystoscopic examination showed the direction of the urethra to be downward and backward rather than in the normal "curve of Carus." The posterior urethra is relaxed and included in a cystocele which is anterior to Mercier's bar. However, on straining, although the trigonal muscle becomes more prominent, the sphincter does not move.

This case had a relaxed internal sphincter and contraction of the trigonal muscle could not

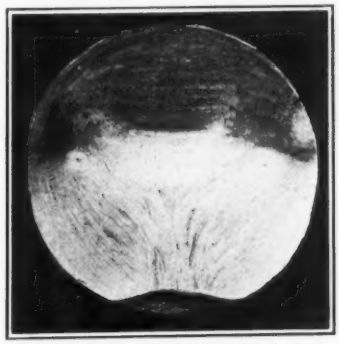

Fig. 11. Case showing general relaxation of bladder.

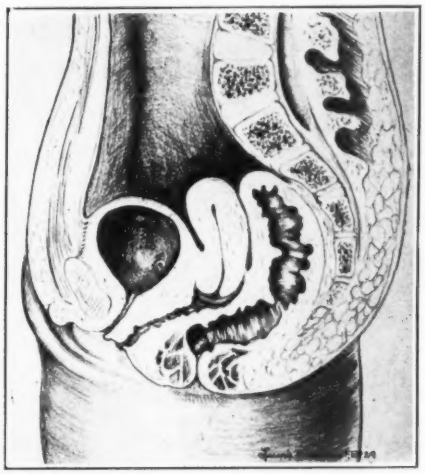

Fig. 12. Sagittal section showing cystocele involving internal sphincter and deep urethra.

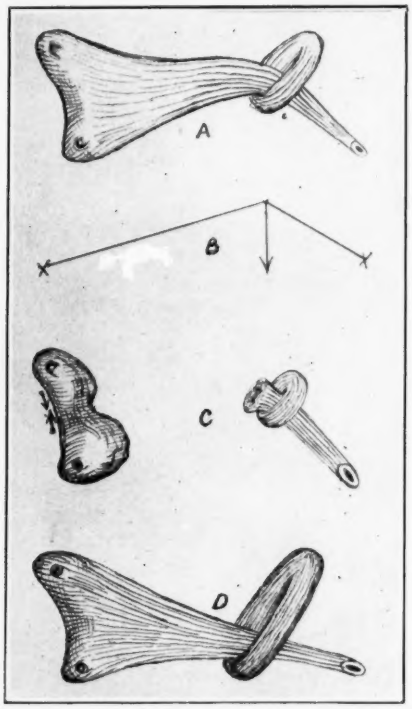

Fig. 13. "A" and "B" schematic drawing from Young's Urology. "C" schematic drawing showing division of trigone muscle. "D" schematic drawing showing trigone in cystocele involving sphincter and causing relaxation. Note contraction of trigone muscle would only shorten urethra.

further depress it because it is a straight pull rather than an angle pull. But it did close the midportion of the urethra as one narrows the lumen of a rubber tube by stretching it. Thus it is easy to understand why it is necessary for the patient to relax before she urinates. A repair of the pelvic fascia in this case will improve the cystocele, but the sphincter may or may not be restored, depending on the ability of the muscle to recover its contractile power.

I believe we should also consider some of the bad results from the present day types of cystocele operations. I shall not consider the accidental passing of a stitch into the bladder nor the production of fistula, of which all are familiar. The most important complications are:

(1) Return of the cystocele.
(2) Overcorrection.
(3) Shortening of the base ot the bladder.
(4) Formation of pseudodiverticula from over shortening the round ligaments beneath the bladder.

(1) *Return of the Cystocele.*—Briefly, in nearly every case the fascia was not sutured anteriorly under the posterior portion of the urethra, leaving the urethrocele uncorrected; or because of injury to the trigone muscle, the increased pressure necessary to open the sphincter separates the suture line.

Case Report.—Mrs. S., age 41, para four, came under observation because of severe bladder tenesmus, passage of blood and pieces of sand. The patient gave a history of repeated attacks of cystitis. The Bovee or uterine shelf operation had beem performed two years previously. The patient believed the bladder irritation had been caused by prolonged use of the indwelling catheter.

Cystoscopic examination showed a small cystocele anterior to Mercier's bar with an area of leukoplakia extending from the ureteral meatus to the internal sphincter. The bladder irritation has been relieved by vesical irrigations with mercurochrome.

I believe this case represents what will occur when the trigone is destroyed and it is necessary to open the internal sphincter by increased vesical tension alone. The uterine shelf or Bovee operation usually gives a firm superior vaginal repair, but it was not sufficient in this case. It should impress on the surgeon the necessity of taking extra precautions where the trigone muscle is destroyed.

(2) *Overcorrection.*—

Case Report.—Mrs. O., age 51, para two, came under observation because of painful urination. Examination showed a large cystocele involving the entire base of the bladder and a large rectocele. She was referred to a surgeon for repair. Six days after operation, I was called to see her because of severe vesical pain and constant desire to urinate. Instillation of sterile mineral oil was given with partial relief. The patient was cystoscoped on the tenth day, at which time the midportion of the base of the bladder was found elevated from the internal sphincter posteriorly well behind Mercier's bar and forming a "basfond" on either side which contained purulent urine.

I have seen this in several types of cystocele operations, but the outcome has ultimately been good, as muscles have stretched, allowing the bladder to assume its normal contour.

In contrast to this case I recently saw a case in which the physician tried to correct the cystocele by plicating the base and posterior wall from above, with a resulting vesical ulcer of nine years' duration.

Case Report.—Mrs. G., age 54, para six, was referred to me because of constant burning and desire to urinate. This was never relieved, but any attempt to retain urine was practically unbearable.

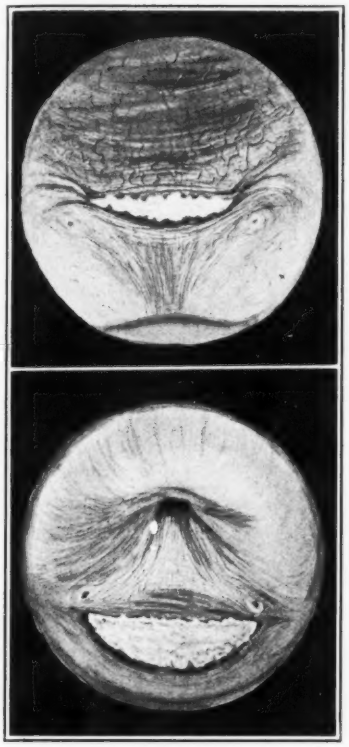

Fig. 14 (top). Ulcer of bladder resulting from folding bladder wall in attempting to cure cystocele.
Fig. 15 (bottom). Suprapubic appearance of same.

The patient states that in 1921 she had a hysterectomy and the physician told her he did more to her bladder than he did to the average case. While in the hospital the bladder pain began and continued with increasing intensity during the past year.

The cystoscopic examination showed two parallel folds just posterior to Mercier's bar with an ulcerated area between. The patient was unable to tolerate distension of the bladder. Under spinal anesthesia I did a suprapubic cystotomy and found an ulcer 1½ by 1 inch with the folds as seen through the cystoscope (*Fig. 13*). This area was resected. The ultimate outcome is, of couse, doubtful as ulcers are prone to recur.

(3) *Shortening of the Base of the Bladder.*—Watkins gave as one of the requirements of a satisfactory cystocele operation, that it should not shorten the vagina.

Case Report.—Mrs. H., age 58, para one, complained of attacks of burning on urination and loss of support to the bladder. This dated back to four years previously, when she felt something "give way" while playing golf. The patient showed a severe perineal laceration with a small cystocele. The Bovee operation was performed April 2, 1927. One year later she was seen, at which time she complained of a dragging feeling in the pelvis which was exaggerated when she sat down, and which compelled her to rest her feet on a chair constantly. Cystoscopic examination one year after operation showed a distinct band across the posterior wall of the bladder, especially when the thighs were extended. The cystocele was removed, but this complication was more disabling than the original complaint.

(4) *Formation of Pseudodiverticula by Overshortening the Round Ligaments Beneath the Base of the Bladder.*

Case Report.—Mrs. C., age 46, para three, was seen one year after a vaginal hysterectomy, because of extreme pain in the bladder and frequency of urination. The patient was unable to retain urine over thirty minutes. She felt that something pulled across her vagina.

The pain was aggravated by sitting down. She had had urethral dilation with no relief.

Cystoscopic examination showed a band running transversely across the base of the bladder, forming a pocket behind, which does not empty, as shown by cystogram. The bladder at the beginning of my observation would hold only 2 ounces.

I have been able to help this patient by using oily preparations in the bladder with over-distension, but hesitate to try to lengthen the pelvic sling.

CONCLUSIONS

(1) In cystocele there is not only separation of the vesical fascia, but often there is also injury of the trigone muscle. A failure to recognize this has often resulted in poor results after a cystocele operation.

(2) Prophylaxis against vesical injury is best accomplished by frequent emptying of the bladder during labor.

(3) The division of the trigone muscle is seldom complete and the remaining muscles might hypertrophy sufficiently to open the sphincter normally if they were not allowed to become stretched by increasing cystocele formation. I am not in a position to say what we could accomplish by using a pessary which supported the base of the bladder.

(4) All medical measures failing, I believe an early repair before the remaining fibers are destroyed by stretching is highly desirable.

(5) If the cystocele is posterior to the trigone the result after suturing the vesical fascia will be good, but if it is anterior to Mercier's bar or if both types are present, the result depends on the power of the remaining trigonal muscle fibers to hypertrophy and open the sphincter normally.

(6) Cases with cystoceles anterior to Mercier's bar are prone to recur because of the increased tension necessary to open the sphincter.

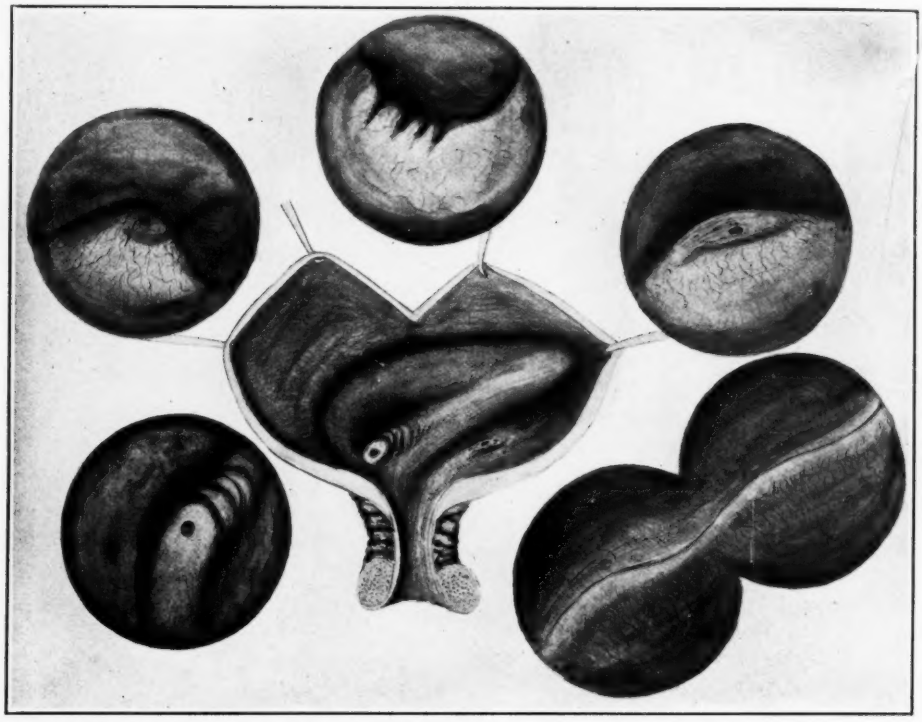

Fig. 16. Case illustrating ridge produced by band from overcorrection in vaginal hysterectomy.

(7) Care should be exercised not to overcorrect the cystocele, as this is often worse than the original condition.

(8) Cooperation between the cystoscopist and surgeon is highly desirable.

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DISCUSSION (*Abstract*)

Dr. H. W. E. Walther, New Orleans, La.—This is rather a common condition seen in routine cystoscopies by urologists.

Women usually come to us as a last resort. In my own experience most of them have been operated upon, either once or twice. In my observations these operations have all been total failures. There seems to be a complete relaxation of all the structures at the external genitals. As soon as you separate the vulva you see the cystoceles. You need not cystoscope to make a diagnosis. But we see a great number of failures following surgery in these cases. We should devise new methods of repair or use the non-operative measures mentioned, particularly over-dilatation of the urethra.

Dr. Joseph H. Smith, Memphis, Tenn.—Cystocele in women is not an uncommon occurrence. It is a wonder to me that women have not more trouble with bladder symptoms on account of this condition. Cystocele, in the majority of cases, in my experience, gives very little discomfort, but occasional cases have a great deal of bladder trouble. Perineal tear with the consequent destruction of the support of the pelvic floor is responsible for most of them. Women with perineal tears develop rectoceles, cystoceles and often a general viscer-optosis, with misplacement of the pelvic and abdominal organs.

Dr. M. M. Coplan, Miami, Fla.—I should like to ask Dr. Van Duzen what conditions he has found of Bell's muscles in those cases where there is a definite stricture in the urethra in the female?

We, of course, have our ideas of the male bladder after long standing strictures of the urethra. The female case presents various symptoms, and I wonder whether there is any atrophy or destruction of Bell's muscles in these cases?

Dr. Frank S. Crockett, Lafayette, Ind.—These cases of cystocele, as they are repaired by the gynecologist, or the abdominal surgeon, leave a good deal to be desired. It seems to me that they really belong to the urologists.

I want to ask Dr. Van Duzen if there is a real destruction or tearing or atony of Bell's muscle that has to be repaired, or whether it is possible to support the bladder, pulling up on the posterior bladder wall, as we attach the bladder to the belly wall in a prostatectomy; or could

the peritoneum be stript off the bladder, allowing the posterior wall to be pulled up, obliterating the cystocele and attaching the bladder to the belly wall. We are not getting a good mechanical repair by the methods which we use now.

Dr. Van Duzen (closing).—I do not do gynecological operations but restrict myself entirely to urology. However, I was connected for nine years with a general clinic and have assisted in many of these operations. Since I began pointing out this injury to the surgeon, or gynecologist, and insisting on plicating the fascia well down under the internal sphincter, the results have been better.

In answer to Dr. Crockett, I wish to point out that when the trigone muscle is torn there is always accompanying separation of vesical fascia. Therefore, if you attempt to cure the cystocele by making the bladder base taut, you have not remedied the separation of the fascia, which is the weak part of the vesical wall and site of future potential hernia or cystocele formation. Such a method would only be temporary at the best.

I believe our greatest help will be from the obstetrician. Frequent emptying of the bladder is necessary during the first stage of labor. Next, if the bladder base is injured during labor, an early repair before these muscles are completely atrophied is desirable. It may be possible to support the bladder after labor by the use of a pessary, but I am unable to discuss it.

In answer to Dr. Coplan, I have found, in women with stricture of the urethra, that the trigone muscle may be hypertrophied or may be atrophied. The power to hypertrophy is more marked in young girls, and in the large atonic bladders we usually see atrophy.

EPIPHYSEAL SEPARATION DUE TO MUSCULAR ACTION: REPORT OF TWO CASES*

By ALPHONSE H. MEYER, M.D., F.A.C.S.,†
Memphis, Tenn.

The separation of an epiphysis through the direct action of a muscle offers an interesting situation, a condition occurring rarely, and for that reason worthy of mention.

It so happens that within the last year I have seen two cases of epiphyses detached through muscle pull. In reviewing the literature upon the subject, I am impressed with the fact that very few cases have been reported. My associate and I are connected with the free services of several hospitals, and reviewing our experiences on the fracture service of these various institutions, and in our private practice as well, all of which covers many hundreds of cases and includes practically every type of fracture, neither of us can recall ever having seen a case, except these two. It has seemed, therefore, worth while reporting these cases, which, from an etiological standpoint, may prove interesting. Certainly after the injury has occurred, there is nothing complicated about the modus operandi of treatment. The interest, therefore, would be centered almost wholly in the causative factor.

In cases upon which this article is based there was a separation of the lesser trochanter of the femur and of the anterior superior spine of the ilium. In neither of these cases was there any external violence, nor was there any evidence of pre-existing disease. Both occurred in youths previously healthy, who have remained healthy since their injuries. The violence was evidently direct insofar as muscular action was concerned.

Broca[1] states that he does not believe muscular contraction sufficient to separate an epiphysis. Scudder[2] says: "Greater force is necessary to cause a separation of an epiphysis than is required to cause a fracture of the same bone." If this be true, for a muscle or group of muscles to be able to produce such a separation, a superhuman muscular effort must be required.

Poland[3] is authority for the following statements. Muscular violence occasionally causes the detachment of one or another of the larger epiphyses of the long bones, but it is the smaller ones, as, for example, the lesser trochanter of the femur, the anterior superior and inferior spines, and so forth, to which powerful muscles are attached, that are most frequently separated by muscular action. He says, further, that formerly it was thought that infancy was the usual time for these injuries to take place, but later experience shows that the most common period for separation to occur is between eleven and eighteen years of age. This author classifies epiphyseal separation into "complete or pure" when it extends through the whole length of the epiphyseal line, and "partial or incomplete" when it proceeds through a certain portion of the line and then finishes its course as a fracture of the diaphysis of the bone.

In the cases reported, the separation of the lesser trochanter offers itself as a case of "complete or pure," while that of the anterior superior spine may be classified as one of "partial or incomplete," inasmuch as the anterior superior spine does not of itself form a separate epiphysis, but is a part of the iliac crest, the whole crest, including the anterior superior spine, representing an epiphyseal unit.

A brief review of the anatomy shows that the lesser trochanter unites with the shaft of the femur about the seventeenth year, and that the epiphysis of the crest of the ilium does not unite with the main portion of the bone until the twentieth to the twenty-fifth year. To the lesser

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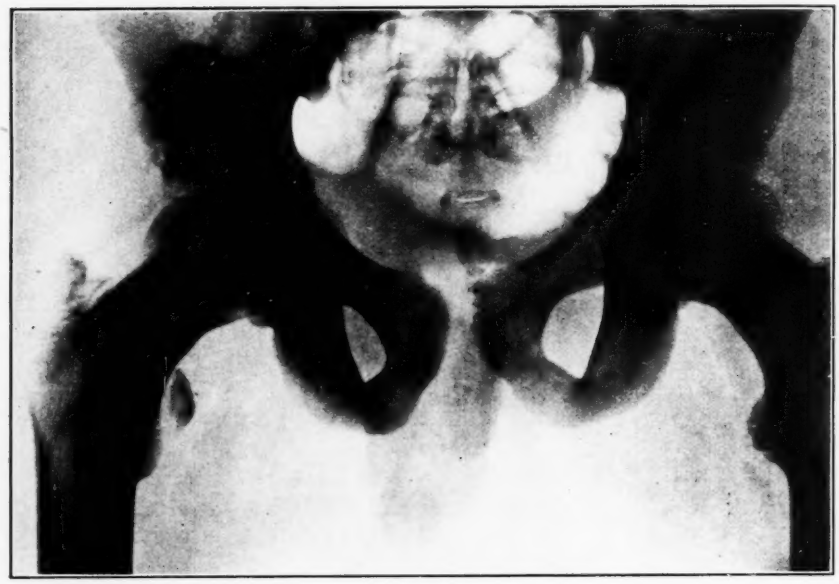

Fig. I, Case 1
Showing separation of the epiphysis of the lesser trochanter of the right femur

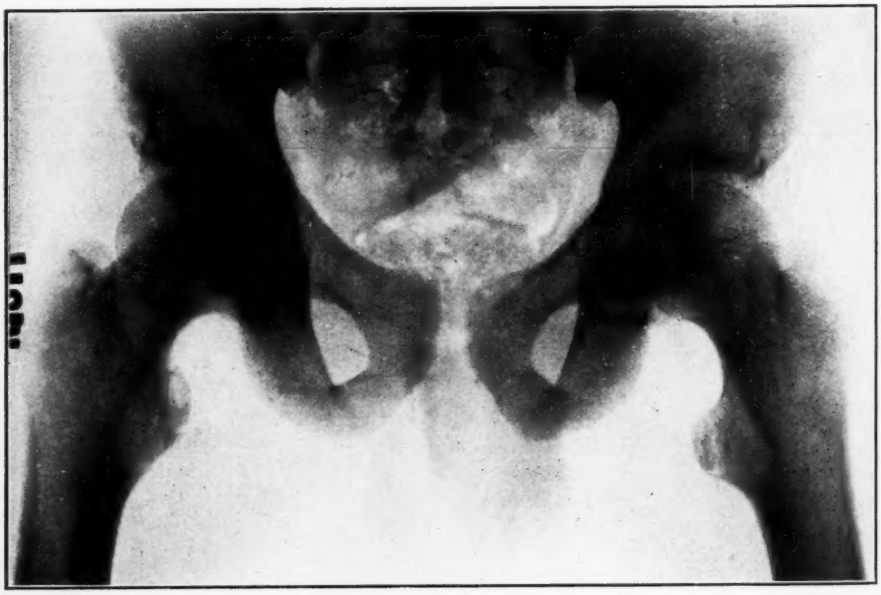

Fig. II, Case 1
Showing the separated epiphysis of the lesser trochanter united to the shaft. (Attached at a slightly higher level than normal.)

trochanter is attached the tendon of the psoas, while several powerful muscles arise from and around the anterior superior spine, namely: the tensor vaginae femoris, the sartorius and the iliacus. These are mentioned merely to refresh the memory, in order to give an idea of how much muscular pull normally is expected upon these parts.

Metcalf[4] reports two cases of separation of the lesser trochanter, one case the result of hyperextension of the thigh, and the other the result of muscular action. This is extremely interesting, as we have here two causes for the same injury, neither of which includes any external violence. Sonnenschien[5] mentions also a case of separation of the lesser trochanter, which occurred in a thirteen-year-old boy while he was running around a track. He considers muscular action responsible for the injury. Schlueter[6] cites a case of a fourteen-year-old boy who had a separation of his lesser trochanter, which occurred during a game of baseball. He says:

"The patient had stopped abruptly at second base, and while attempting to keep his body upright, experienced a sudden severe pain in the left hip, which forced him to lie down." Schlueter states that: "Strong muscular contractions not uncommonly produce fractures of bone. An avulsion of the lesser trochanter of the femur belongs to this class."

I have been able to find reported only one case of epiphyseal separation of the anterior superior spine. If other cases have been reported in the literature and were overlooked, then I offer my apologies to the authors.

Akin and Carlson[7] state that a boy "aged 15 . . . having covered half the course in a 100-yard dash, felt a sudden pain and 'something give way' in his right side. He finished the race and collapsed." Subsequent examination proved that he had an epiphyseal separation of the anterior superior spine of the ilium. They gave as the etiological factor, muscular violence.

Each of the authors quoted, in turn, mentions the rarity of this lesion, and our own experience certainly coincides with theirs. The lesion is surely unique and unusual.

REPORT OF CASES

Case 1.—R. H., age 14, entered the hospital on April 16, 1929, giving the following history: The day before, while at school, he and his classmates were jumping over a pole about three feet from the ground. He had jumped the pole one time without any ill effects, but upon his second attempt, just after he had left the ground, he was seized with a sudden acute pain in the region of the right groin, which caused him to fall to the ground. For the moment he was unable to get up or walk and was carried into the school house. Later, however, he walked home, but had considerable pain and was barely able to reach his home. He remained at home over night, without any medical attention, but suffered throughout the entire night with pain centered over the region of the upper part of the right thigh. Upon admission to the hospital, examination revealed swelling in the upper part of the right thigh and exquisite tenderness over the region of the lesser trochanter. He was unable to flex the thigh, and hip joint motion caused pain. There was no external rotation of the limb, nor was there any shortening.

Family history and past history were unimportant. The blood Wassermann was negative.

X-ray pictures revealed a complete separation of the epiphysis of the right lesser trochanter, with upward displacement. On April 18, a plaster spica was applied extending from the costal margin above to the toes on the affected side, and to the knee on the uninjured extremity. The thigh was flexed to an angle of about 135 degrees, and the knee also flexed. Subsequent x-ray pictures made through the cast showed the detached fragment to be in good position, riding only slightly above its normal attachment.

At the end of the fifth week, the long spica was removed, the thigh extended to about 160 degrees, and a short hip spica applied extending to the knee only. This cast was left on for three weeks, at the end of which time it was removed and in a few days the patient was allowed to be up and about on crutches.

X-ray pictures following the removal of the last cast showed perfect union of the epiphysis at a very slightly higher level than the normal.

Fig. III, Case 2
Showing epiphyseal separation of the anterior superior spine of the left ilium.

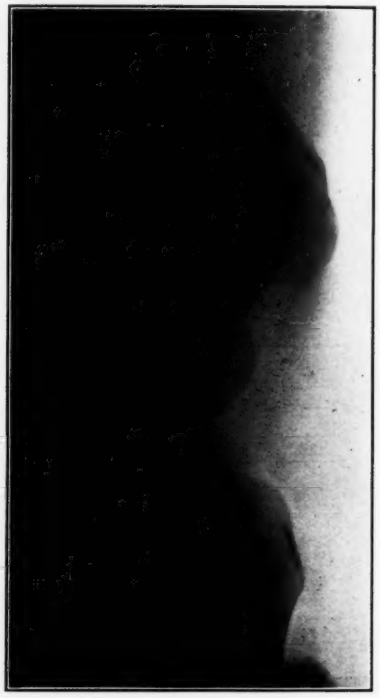

Fig. IV. Case 2
Showing the separated anterior superior spine firmly united to the ilium.

Two weeks later crutches were discarded and weight bearing permitted.

Complete functional recovery was obtained.

Case 2.—E. T., age 17, on May 4, 1928, while running in an interscholastic track meet, at a fairly good pace, but not sprinting, felt a sudden sharp pain in his left side, and after taking a step or two, fell to the ground. He was unable to walk and had to be carried.

The family and past history were negative.

When he was first seen his pain was directed to the left anterior superior spine. He was unable to flex his thigh and the extremity was rotated externally. A freely movable mass was palpable just below the normal level of the anterior superior spine and clinically it was self-evident that the anterior superior spine had been detached from its mooring.

X-ray confirmed the diagnosis.

On May 5, under general anesthesia, the anterior superior spine was easily replaced by flexing both the thigh and the knee, which, of course, completely relaxed the sartorious and iliacus muscles. A plaster hip spica was applied to the ankle with the extremity held in this position. It was carried to the knee on the opposite side.

June 11 (thirty-seven days later) the long spica was removed and the thigh nearly completely extended. A short hip spica was applied as far as the knee.

X-ray pictures made between casts showed union of the anterior superior spine and good position.

The short spica was left on until July 3, which was approximately two months from the date of the application of the first cast. After removal of this cast, subsequent pictures showed excellent healing and position.

On July 28, he was discharged as having recovered completely, both anatomically and functionally.

COMMENT

Cases of epiphyseal separation, due to muscular action, offer an interesting study. It is certainly an unusual phenomenon, and perhaps a physiological cause other than a supreme muscular effort might be entertained as an etiological factor. Is it possible that there is a sudden incoordination between the antagonistic groups of muscles which causes an undue strain upon the affected epiphysis? At any rate, there is surely food for reflection which allows the imagination a wonderful opportunity, and there is plenty of scope for subsequent investigation.

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DISCUSSION (*Abstract*)

Dr. Guy A. Caldwell, Shreveport, La.—A few days after I received Dr. Meyer's paper, I saw a report of a case of this kind in the *Journal of Bone and Joint Surgery*, (11:876, Oct., 1929), which I am going to give. It was on "Epiphyseal Separation of the Trochanter Minor."

Dr. Paul Lapidus presented the case of a colored boy about fourteen years of age, who, while running, felt severe pain in his groin and was hardly able to walk home. He was obliged to stay in bed for a few days and was seen in the City Hospital.

The most peculiar point was the gait which showed a deficiency of flexion of the thigh. While walking, in order to flex the thigh, the boy would suddenly swing his body backward so as to throw the thigh forward. This peculiar gait, together with the considerable tenderness which was strictly localized over the smaller trochanter, helped us to make the proper diagnosis before the x-ray was taken. The Ludloff sign in this case was negative, as the patient was unable to

lift the heel while lying in the prone position on his back. While sitting he was able to flex his thigh slightly, using as flexors of the thigh the rectus femoris muscle and the tensor fascia lata and sartorius.

So far, in the literature up to last year, there were reported about forty-five cases, in most of which the diagnosis was not made at all and the condition was accidentally discovered by x-ray.

This report does not go into the treatment of these cases. Dr. Meyer's procedure was evidently correct, since the end result was excellent.

Dr. Donald T. Babcock, Miami, Fla.—We had recently a case which resembled the ones mentioned, but it turned out to be of another kind.

A young football player, age sixteen, while running for a touchdown, felt a sudden pain in the right side. Six hours later, he had a swelling just below the anterior superior spine on the right side. X-ray films were negative for separation of the epiphysis. He was immobilized in plaster spica for about two weeks and cleared up very nicely.

Dr. Meyer (closing).—There may be other causative factors than muscle pull. Perhaps there is a sudden incoordination between antagonistic groups of muscles which causes the tearing off of an epiphysis. As to the anterior superior spine of the ilium, I have seen nothing in the literature as to the whys or wherefores of its slipping. With reference to the lesser trochanter of the femur, Binet and Hamant state: "In case the body is semi-flexed at the hip joints with the ilio-psoas muscle contracted and pulling perpendicularly, a sudden effort to stand upright will put an undue strain on the small trochanter."

CHRONIC APPENDICITIS: DIAGNOSIS AND SIGNIFICANCE*

By THOMAS R. BROWN, M.D.,
and
ERNEST H. GAITHER, M.D.,
Baltimore, Md.

The term, chronic appendicitis, is a much abused and disputed one; in fact, various surgeons and internists deny that such an entity exists. One must admit that in a certain number of cases they appear to be justified in this stand, since the diagnosis of chronic appendicitis has frequently been misapplied to a syndrome simulating it, but actually deriving from a low-grade inflammation involving the terminal ileum, cecum, and ascending colon, usually associated with atonic and visceroptotic conditions. In these cases the patient's complaints are intensified by an appendectomy.

There is, however, a definite number of cases of true subacute or chronic appendicitis in which it is impossible to secure a history of acute local outbreaks. Other patients present a history of acute attacks in a past so distant that the local manifestations, if any were present, have been forgotten. At a later date, however, mild or severe symptoms may be referred to the epigastric region, producing a hyperacid, pylorospasm syndrome suggestive of ulcer or disease of the right upper quadrant. The diagnosis of these cases is difficult and the treatment peculiarly unsatisfactory with symptomatic therapy, such as rest, local applications, smooth, non-irritating diet, sedatives, anti-spasmodics, and alkalies. Unquestionably this unsatisfactory result is due to the fact that the underlying pathological condition is a diseased appendix which has not been recognized and appropriately treated.

A detailed discussion of the various types of chronic appendicitis with their multiplicity of variable symptoms, which shows the protean aspects of this widespread and often unrecognizable malady, will be found in a paper published by one[1] of us some years ago.

We now present the results of our studies to date: a group of cases of true chronic appendicitis in which our diagnosis has been greatly facilitated by careful fluoroscopic study extending over a number of days, and by placing the patient before the fluoroscope in various positions, with close observation of the lower right quadrant area before and after active purgation. Before detailing the method, report of cases, findings, and conclusions, we shall discuss briefly the views we hold regarding the entity, chronic appendicitis.

Because of the bizarre and constantly changing picture or syndrome which mirrors the results of an underlying pathological appendix, great difficulty is at times encountered in evaluating and interpreting properly the subjective symptoms and objective signs; for one must remember that this pathological entity often changes symptoms with chameleon-like swiftness. The symptoms may be permanent, though more often fleeting; they may be mild or severe; they may assume the characteristics of any of a dozen pathological conditions: gastric or duodenal ulcer, hepatobiliary disease (cholecystitis, cholelithiasis, cholangitis, duodenitis); nephrolithiasis, sacroiliac or lumbar muscle strain, functional dyspepsia, or a digestive neurosis. Again, a pathological appendix may be the true underlying

*Read in Section on Medicine, Southern Medical Association, Twenty-Third Annual Meeting, Miami, Florida, November 19-22, 1929.

1. Gaither, Ernest H.: Chronic Appendicitis. S.M.J., March, 1921.

factor of a mucous colitis, though involving only the descending colon or sigmoid with a severe colonic spasticity; or a circumscribed pathological entity such as typhlitis or perityphlitis may be the end product of an advanced and prolonged appendiceal disease. But the mere fact that these subjective symptoms and objective signs are not clear-cut and decisive is no valid reason for dogmatically denying the existence of the entity, although there are those who would have us believe that chronic inflammation of the appendix and its closely related structures is a myth, and the diagnosis, chronic appendicitis, consequently without sound basis.

After years of close study of subjective symptoms, objective signs, biopsy, and the continued preoperative and postoperative observation of numerous cases, we are more than ever thoroughly convinced that the pathological entity, chronic inflammation of the appendix, does exist.

We are well aware that inflammatory reaction and structural tissue changes are not always confined to the appendix. However, the fact that surrounding structures are involved is certainly no proof that the appendix was not the primary site of infection and inflammation, and that the disease of the surrounding tissues was not secondary to that infection. At this point it will be interesting and illuminating to quote Braithwaite's theory, which we believe accurately meets many instances:

> "What is it that takes place in an attack of acute appendicitis? There is poured out a great quantity of fluid, at first of bactericidal potency but quickly becoming contaminated. The results of the subsequent inflammation are not confined to the appendix itself, but are frequently many inches away from it, as is shown by bands of adhesions in the neighborhood. If this be true of acute appendicitis, may it not be true to a lesser extent of the subacute or even chronic variety?"

Let us here briefly note a finding which we have proven to be of valuable assistance in the diagnosis of chronic appendicitis; this is, the occasional discovery, sometimes after marked physical exertion on the part of the patient, of a moderate leucocytosis, or a slight rise of temperature, or both. In the majority of cases such a condition will not be noted; however, when it is observed, its possible significance should receive consideration.

In our opinion, it is entirely possible for lower right quadrant disease to manifest itself exclusively in epigastric and upper abdominal symptoms. Therefore, at this time, we desire to call to your attention with particular emphasis, our experience regarding pyloric-duodenal symptoms in cases of chronic appendicitis.

It is a not uncommon experience to meet with patients whose histories present symptoms which are definitely typical of peptic ulcer in the pyloric-duodenal area: that is, an early history of irregular and indefinite dyspeptic symptoms noted in the epigastric area, which later develop into a fixed syndrome presenting the well-known and classical subjective symptoms and objective signs: pain, usually of a dull, gnawing, weakening type (although it may present other characteristics), located in the epigastrium. Unless complications have arisen, it is in most instances localized in this area. There is a definite meal relation, and food ease, as characterized by complete relief or marked alleviation of pain after food intake; also the well-known nocturnal attacks, for which the patient provides by having at the bedside the proverbial cracker, milk, or sodium bicarbonate. Further, a history of chronicity, intermittency, and remittency is obtained; also a history of nausea, and at times, vomiting; indeed, hematemesis in these cases is not unknown. Localized epigastric tenderness and soreness are often emphasized, and in many cases there are no subjective symptoms to indicate localization of the pathological condition in the appendix, as all symptoms are referred to the upper abdomen, due to impulses carried through the vegetative nervous system, which bring about inhibition of pyloric relaxation, intensification of peristalsis, rise of intra-gastric pressure, hyperchylia gastrica, and hyperchlorhydria. In short, a subjective syndrome is produced which is in every detail symptomatic of an ulcerative lesion in the pyloric-duodenal area. Of course, at times, we may have to deal with a double lesion, a chronic appendicitis accompanied by a duodenal or gastric ulcer.

These enumerations represent the outstanding subjective symptoms, general and specific, as revealed in the history presented by the patients.

Objective findings may be equally misleading, and such symptoms as vomiting, epigastric muscular resistance, hyperchylia gastrica and hyperchlorhydria, may be presented as prominent complaints.

The x-ray, most helpful in many instances, may in some cases divert us from consideration of the primary seat of disease because of gastric hypertonicity, pylorospasm, and hyperperistalsis. Other objective radiographic signs which may lead one astray are indentations on either curva-

ture of the stomach, or surfaces of the duodenum, due to spasm, and distinctly persistent. In many such instances they disappear only after thorough saturation with atropine or belladonna.

Persistent or intermittent occult blood in the stool is often puzzling and misleading, and will aid in diverting us from a correct diagnosis if we are not extremely cautious.

We have found it very necessary to exercise great care in our study and observation of the subjective symptoms and objective signs of these cases in order to avoid the pitfalls which beset the path to correct conclusions and diagnosis.

We desire at this moment to present the method which we are finding most helpful as an adjunct in the diagnosis of pathological appendices.

Our method of investigation is as follows: four ounces of barium sulphate in one pint of water are administered eighteen hours before the first fluoroscopic investigation, at which time we place the patient before the screen in the recumbent posture. Having made a thorough examination of the right lower quadrant area and the colon, we prescribe a potent saline to induce a thorough evacuation of the colon before proceeding with further observation of the appendix. At both primary and subsequent studies, we make various notations as follows: we first note the position, tone, and any abnormality of the colon; after which we divert our attention to the right lower quadrant in an endeavor to discover a visualized appendix. If this is observed, particular attention is paid to its length, its direction, possibility of displacement by manipulation, and the reaction to manipulation. It is most interesting to note the change in shape often brought about by palpation, due to the contraction and relaxation of the musculature of the appendix. Interest has on several occasions been aroused by a curled appendix which could be straightened out by deep palpation and manipulation, but which in a few moments would display vermiform movements, and a return to its original contour. In a surprising number of instances the organ presents a segmented or beaded appearance, or one portion may be quite constricted while another is markedly dilated. Again, we have noted a decided narrowing to within 1 or 2 cm. of the tip, at which point it was quite bulbous. In such cases it has been significant to us to have the surgeon discover at operation a fecal concretion quite frequently permeated by barium. In certain cases there was a filling of the proximal portion only, followed, after a laxative, by a visualization of the entire organ. Again, there would be visualized only a bulbous tip with a narrow barium outlined lumen, although sometimes no barium outline was apparent.

Having thus studied the morphologic characteristics of the appendix, we next note the results of palpation as regards the sensory response, and we find these most impressive. We believe it is valuable to note, after repeated palpation, a circumscribed area or areas of tenderness. Direct pressure on the appendix will in most instances cause distinct discomfort or pain, and thus far more sensitiveness has been noted from the tip to its cecal attachment than at the ileocecal junction and McBurney's point. An observation of the greatest interest is that in a small but definite number of cases, in which the appendix was not located at McBurney's point, more exquisite tenderness was elicited by palpation at McBurney's point than was elicited by pressure applied to the visualized appendix. This extremely interesting note must, however, be emphasized as the exception and not the rule. It is important to state that pressure upon the ileocecal junction and McBurney's point, in cases harboring a chronically inflamed appendix, will usually produce local pain or discomfort. During the process of palpation we particularly noted the relation of deep palpation to referred pain, and observed that in the majority of cases moderate or marked discomfort, at times amounting to true pain, was produced in the epigastric and left lower quadrant areas; indeed, in a few instances, left upper quadrant distress was also noted. Also of interest were those cases in which deep lower right quadrant palpation failed to produce local discomfort, but did bring about distress in the upper or lower left abdomen. In a few cases of operatively demonstrated disease of the appendix neither local nor reflex pain nor discomfort was produced by deep palpation, this in the face of outstanding subjective symptoms. In the majority of cases, the patients volunteered the information regarding referred discomfort or pain.

The following observation was made in one of our recent cases, in which the appendix was visualized, and the clinical history was indicative of a pathological process in this organ:

When deep pressure was applied from the tip of the appendix to the junction with the cecum, very marked local sensitiveness and epigastric discomfort were noted. Immediately following this procedure, palpation was

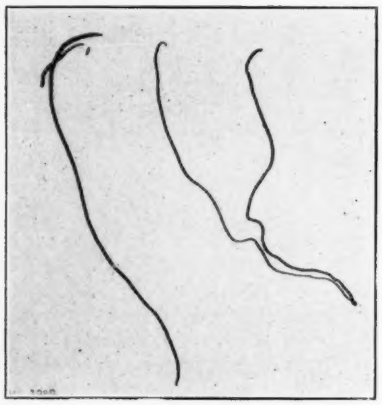

Fig. 1
Visualized appendix tending toward segmentation; constricted type.

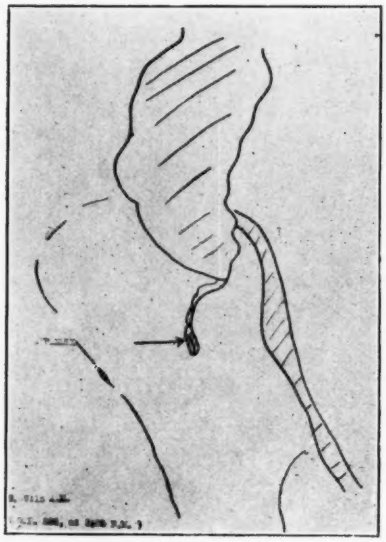

Fig. 2
Segmented, bulbous appendix and visualized terminal ileum.

likewise practised upon the ileocecal junction, and McBurney's point, and the patient voluntarily laid most striking emphasis upon the difference in severity of local and reflex pain and discomfort, that caused by palpation of the appendix being most marked. Four days after the first examination, when the large intestine was free, but the appendix was still visualized, pain, both local and reflex was produced by pressure upon the appendix, while palpation at the ileocecal junction and McBurney's point failed to elicit reflex discomfort. Subsequent reports from the surgeon and the pathologist proved the preoperative diagnosis of chronic appendicitis correct.

Cases have been observed wherein localized pain has been noted only upon palpation of the appendix, and no reflex discomfort elicited. In a few cases, pressure at the ileocecal junction and at McBurney's point caused neither local nor reflex sensitiveness.

After an intensive study of the clinical history, physical examination, preoperative and postoperative observations, we are thoroughly convinced that circumscribed, localized, and referred pain, produced by palpation in various areas of the lower right quadrant, has true diagnostic value.

It should be noted that in the majority of cases presenting a pathologic appendix which is visualized there is an accompanying overnight ileal stasis.

In this study, particular attention has been paid to the tone of the colon; and we are prepared to say that in the greater number spasticity to a marked degree was noted, and that in a certain number, palpation along the course of the large intestine was productive of discomfort in varying degrees. Spasticity and hyperperistalsis of the pylorus were noted quite often, but were not so frequently observed as the spastic colon.

Fig. 3
Figure 8 appendix verified by operation.

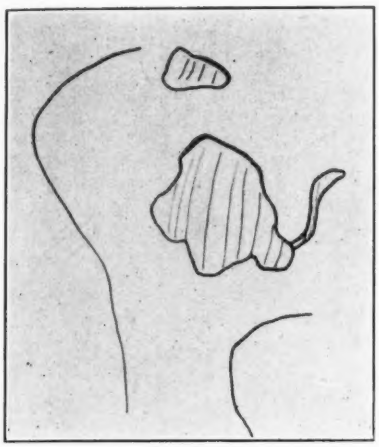

Fig. 4
Fluoroscopic tracing. Artist's drawing, Fig. 5, illustrates this.

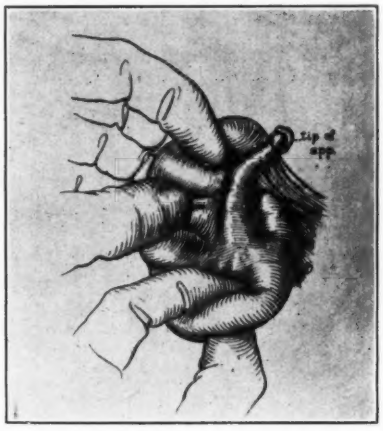

Fig. 5
Drawing at operation. Confirms Fig. 4.

The filling and emptying of the appendix has been of genuine interest in a number of cases. Toward the end of the first twenty-four hours, 1 or possibly 2 cm. of the appendix would be visualized. After purgation, and during the succeeding days, the full length would be visible, followed by a gradual elimination of the barium. This method of observation certainly bids fair to give us an idea as to the motor capacity of the appendix.

Thus far we have accumulated in this series 114 cases in which the appendix was visualized, and of this number 47 have been operated upon with verification of the diagnosis.

As evidencing the accuracy with which observations have been made by the method described above, we shall very briefly quote the essential important points of the history and preoperative notes made by us in recent cases, and the postoperative reports of the surgeons.

CASES

Case 1.—Mrs. G., a white woman, married, age 32 years, complained of abdominal pain, and of obstinate constipation. For a year she had been suffering with pain in various portions of the abdomen, which at times radiated to the back. The food relation was indistinct. The cardinal symptoms of ulcers, meal relation, and food ease were absent. There was nausea and vomiting at irregular times with no relief. There was much "gas" accompanied by pressure throughout the abdomen; and epigastric soreness and tenderness were frequently noted. The pain which was first noted in the pit of the stomach had spread to the lower abdomen, particularly in the lower right quadrant, and had recently been noted in the lower back (lumbo-sacral area). There was soreness without tenderness in the epigastrium and lower right quadrant.

Physical Examination.—Tenderness of the lower right quadrant, with particular sensitiveness from the tip to the base of the appendix, also at the proximal portion of the cecum. Reflex abdominal discomfort was absent.

A test breakfast showed free acid, 56, and total acid, 74. There were 8,500 to 8,600 white blood cells.

Barium was administered 18 hours before fluoroscopic examination. There was very marked overnight ileal stasis, a long, bulbous appendix, tenderness at the site of the ileocecal junction and also upon pressure upon the appendix. The diagnosis was chronic appendicitis.

Dr. Alfred Ullman, who operated October 26, 1928, reported: "The appendix is 6 cm. long and bulbous at

Fig. 6, Case IV
Cecum rotated.

the tip. There is a calculus three-quarters of an inch long at the terminal portion. The appendix is free in the pelvis. The diagnosis was chronic appendicitis with a fecalith."

The pathologist reported: "The specimen consists of an appendix 10 cm. in length. The mid-portion is dilated to about three times the diameter of the remainder of the appendix. The tip is bulbous and there is a constriction proximal to the tip. The dilated portion is filled by a concretion measuring 2.5 cm. in length and 7 mm. in diameter. On section this is fairly soft and the interior is made up mainly of white material, through which there are veins of brownish material definitely fecal in color. There is marked fibrosis of the submucosa and moderate fibrosis of the muscularis; there is moderate chronic inflammatory infiltration throughout the muscularis. The diagnosis is chronic appendicitis."

Case II.—Mrs. Q. was a white woman, aged 38, married. For fourteen years she had had intermittent abdominal cramps, accompanied by much bloating. The pain was usually general. However, it was often localized in the lower right quadrant. Nervousness seemed to precipitate an attack which might last for several weeks. There was much abdominal pain after defecation, also after food, no matter what its character. During the winter of 1928 she had persistent cramps and tenderness in the lower right quadrant, and in the summer of the same year, hyperacidity and regurgitation of food. Obstinate constipation had been noted for years. However, mucus had never been noted.

Physical Examination.—Palpation of the lower right quadrant produced local resistance and tenderness; also left lower quadrant sensitiveness.

A test breakfast showed free acid, 42; total acid, 66. The white cells were 6,300 to 6,500.

Radiographs in March, 1928, revealed the appendix visualized twenty-four hours after the administration of barium, and empty after seventy-two hours.

Fluoroscopic examination July 1, 1929 (barium having been administered 18 hours previously), revealed a spastic colon with overnight ileal stasis. The appendix was outlined, was moderately curved, somewhat segmented, about 2.5 cm. in length. Pressure upon the appendix and ileocecal junction caused localized tenderness, but no referred discomfort. Pressure along the course of the cecum and ascending colon produced moderate discomfort, but not nearly so marked as when the ileocecal junction and the appendix were palpated. The appendix was visible at the end of 48 hours, but not at 96 hours.

Dr. Thomas S. Cullen, who operated October 16, 1929, reported: "The appendix was embedded in adhesions, and was about 9 mm. across for at least 3 cm. It then became much smaller, and near its tip was about 3 mm. in diameter. The tip was almost completely nipped off. The appendix was adherent throughout its entire extent, and was removed with considerable difficulty. The cecum was tied down to the brim of the pelvis by nonvascular adhesions. An artery forceps could be readily carried beneath these adhesions."

Case III.—Mrs. K., a white woman, age 26, married, complained of chronic constipation, with no mucus. She had generalized abdominal cramps, discomfort and distension, burning back of the sternum, which had been intermittent and remittent during the previous two years. Intermittent mid-abdominal cramps and "gas" were sufficiently severe to cause the patient to stop if walking, and bend over until the attack disappeared.

Abdominal examination revealed a spastic, moderately sensitive sigmoid, and very marked tenderness with muscle spasm at McBurney's point. There was no reflex pain. A test breakfast showed free acid, 32; total acid, 50. There were 5,000 to 9,000 white cells.

Fluoroscopic examination, December 13, 1928, showed a decided overnight ileal stasis. The appendix was visualized, being quite long and segmented. Pressure upon the appendix caused much local discomfort and resistance. The reflex epigastric discomfort caused by pressure directly upon the appendix was marked.

Upon fluoroscopic examination December 17, 1928, following thorough purgation, the colon was empty. However, the barium in the appendix was 2 cm. in length and the bulbous tip of the appendix was clearly outlined.

Upon fluoroscopic examination July 8, 1929 (barium administered 18 hours previously), the appendix outlined was about 2½ inches in length, not curled, and hanging down in the pelvis.

Upon fluoroscopic examination July 10, 1929, the appendix was easily visualized. All barium from other portions of the intestine had disappeared. The appendix was kinked near its base and was segmented; the kinking could be eliminated by palpation.

Dr. William F. Rienhoff, who operated August 7, 1929, reported: "The appendix was found to be twice its normal size; in length it measured about 10 cm. The blood vessels under the peritoneum and over the appendix were very much dilated and there were several bulbous expansions of which the most prominent was at the tip. There were film-like adhesions which bound the appendix down towards the pelvis and which also held its proximal portion in close adherence to the posterior abdominal wall. On opening the appendix after operation it was found to contain two fecaliths the size of small date seeds and similar in shape. The mucous membranes were definitely infiltrated as were the submucosa and the mucosa. The wall of the appendix was very much increased in thickness."

Microscopic examination showed definite chronic appendicitis.

Case IV.—Mr. D., a white man, age 45, widower, for 18 years had had pain in the upper abdomen, accompanied by discomfort, distress and distension after meals, either immediately or in a few hours. At times there was marked nausea. Usually the pain and discomfort appeared quite suddenly. However, they disappeared gradually. There was much gas with sour and bitter eructation. Flatus was marked. It passed easily, with decided relief. At irregular intervals, pain in the lower right quadrant was noted, which was intensified by bending over and by heavy lifting.

Physical examination revealed moderate tenderness with no true muscle spasm, in the right lower quadrant.

A test breakfast showed free acid, 20; total acid, 38. The white cells were 6,400 to 11,200.

Fluoroscopic examination April 24, 1929 (barium administered 18 hours previously), revealed a spastic colon and overnight ileal stasis. The appendix was not visualized. A laxative was administered the evening of April 24.

Fluoroscopic examination, April 25, showed the appendix curled up, kinked, and beautifully visualized.

Pressure on it caused generalized abdominal discomfort, also local sensitiveness.

Upon fluoroscopic examination, April 29, the appendix was outlined and curled up, resembling an S or a figure 8. Deep palpation caused localized tenderness and reflex generalized abdominal discomfort.

Dr. William F. Rienhoff operated May 22, 1929. With great difficulty the cecum was delivered and the appendix brought into the wound. The appendix was bound down with adhesions, and on excision was found to be curved almost into the figure 8. The appendix was so firmly bound down and difficult of removal that at one time it was thought that it would be necessary to make a separate incision for its removal. However, after great difficulty it was delivered into the wound.

Case V.—Miss M., a white woman, 20 years old, unmarried, complained merely of constipation and regurgitation of acid fluids after meals.

Physical Examination.—The cecum and ascending colon were palpable. There was no evidence of muscle spasm nor tumor mass. No pain nor discomfort was caused by palpation.

The test breakfast showed free acid, 52; total acid, 70. There were 8,450 white cells.

Fluoroscopic examination showed a perfectly normal stomach. For three days the appendix was visualized, rather faint, but definitely present. Seventy-two hours after purgation it was segmented. There was some tenderness at the tip on palpation.

Dr. Stuart McGuire, of Richmond, Virginia, operated April 18, 1928. He reported: "A long, large, markedly inflamed appendix, containing several fecaliths."

Case VI.—Mr. J., a white man, age 22, unmarried, two months previously noted epigastric pain, which was almost constant for two weeks. Food would occasionally lessen the pain or completely relieve it. Regurgitation of sour and bitter material often occurred. Soda at times gave him temporary relief, while on other occasions painful distension and gas followed its use. The patient had a three-week period of absolute freedom from the above complaints. However, during the preceding two weeks he had experienced almost constant pain just as noted in the beginning of the attack. He had had attacks of sharp pain in the lower right quadrant at irregular intervals during the previous few years, which would cause him to stop walking for a few minutes. He had no nausea.

Abdominal examination was negative.

A test breakfast showed free acid, 64; total acid, 82. There were 5,000 to 10,000 white cells.

Fluoroscopic examination February 16, 1928 (barium administered February 15), showed no overnight ileal stasis nor cecal dilatation. In the upright position the appendix was clearly outlined, hanging down in the pelvis, thick, and kinked upon itself. In the recumbent posture, the appendix was also clearly visualized. It was kinked and curled upon itself, and toward the tip there was a distinct constriction. Upon pressure the appendix could be straightened out. However, when the hand was withdrawn, it again curled upon itself. Tenderness was not elicited by deep palpation upon the appendix.

Upon fluoroscopic examination March 29, 1928 (barium administered March 28), the appendix was clearly visualized. The cecum and ascending colon were empty; the appendix was segmented and curled upon itself; it could be straightened out easily and there was no localized tenderness. After two or three palpations during which time the appendix was straightened, it was observed that it coiled upon itself and assumed a figure 8 appearance.

Dr. William E. Johnston, of Detroit, Michigan, who operated September 6, 1928, reported: "The appendix was retrocecal, about 10 cm. in length, firm and thickened. It was kinked about 3 cm. from its base. There were adhesion bands from the appendix to the cecum."

The microscopic diagnosis was "subacute appendicitis with partial obstruction of the lumen."

CONCLUSION

Let it be thoroughly understood that we do not, by any means, assert that the mere visualization of an appendix is evidence of pathology. However, we do affirm that, given the findings as above enumerated, plus the clinical history, subjective symptoms, and objective findings (physical and laboratory), we possess in this procedure a truly helpful method. These assertions we make only after continued personal observation and close study of our cases, throughout preoperative and postoperative phases in many instances. In others, through the application of indicated therapy, decided relief has been obtained; and in some cases, complete alleviation of symptoms has been achieved.

DISCUSSION (*Abstract*)

Dr. Julius Friedenwald, Baltimore, Md.—How often do we observe cases in which the appendix has been removed for so-called chronic appendicitis without affording the slightest relief. This is unquestionably due to the fact that other conditions are often at hand producing the symptoms, the result either of widespread disturbances involving other abdominal organs, and not limited to the appendix itself, or of forms of neuralgia occurring in the abdominal wall. The failures in diagnosis are not infrequently due, according to our experience, to imperfect or insufficient clinical observation. It is a well established fact that the clinical signs of chronic appendicitis are rarely distinctive. The lower right quadrant tenderness and pain which are usually held as significant signs of this disease may be due to many other causes and should not be regarded as of great value, unless a history of preceding acute or recurring attacks can be elicited. The gastric symptoms frequently noted are due to pylorospasm, are not infrequently observed in other affections, and are by no means characteristic of chronic appendiceal disease. On the other hand, many of the symptoms apparently caused by chronic changes in the appendix are the result of a general constitutional asthenia not uncommonly associated with visceroptosis, mucous colitis and ileal stasis. In some instances, they may even be due to peptic ulcer or gall bladder disease, affections with which appendicitis is not uncommonly associated. The method suggested by Carnett of differentiating intra-abdominal and parietal tenderness should always be used in the diagnosis and has been of much value in the study of our cases. The Roentgen ray signs are usually misleading and often difficult of interpretation. This method

of investigation is so extremely delicate that many unsuspected findings are frequently revealed which in themselves have but slight clinical significance. These signs can therefore be utilized only as confirmatory evidence of chronic appendicitis, and should be considered of value only when considered with the clinical manifestations of the disease. However, the x-ray may be of unquestionable value in the diagnosis in eliminating the presence of other affections. In a series of 100 cases in which appendectomies had been performed for so-called chronic appendicitis with unsuccessful results, the following diagnoses were finally established:

Peptic ulcer	24
Cholecystitis and cholelithiasis	21
Renal and ureteral diseases	4
Pelvic disease	2
Colitis	11
Neuralgia of the abdominal wall	14
Carcinoma of the cecum	2
Tuberculosis of the cecum	1
Abdominal adhesions	19
Hernia	2
Total	100

On the other hand, in a similar number of instances in which appendectomies were performed for this condition, the results obtained proved most satisfactory. It is quite evident, therefore, that the diagnosis of so-called chronic appendicitis is fraught with considerable difficulty and final conclusions as to the proper procedure to be undertaken can be reached only following a most careful and intensive study of the individual case.

Dr. T. Z. Cason, Jacksonville, Fla.—The question of what constitutes a chronic appendix is in my opinion still unsettled. Undoubtedly the recurrent, acute or even subacute appendix is a chronic one and the advisability of its removal is not debatable. Is there such a condition as a chronic appendix when there has not been an acute or subacute attack? Then, too, is it not true that it is practically impossible for the pathologist to demonstrate a normal appendix removed from any individual past forty years of age, The question is not so much whether there is a disease entity, chronic appendicitis, as when is the chronic appendix offending? A failure to answer this correctly has brought the subject into bad repute and, as the essayists have indicated, caused a serious doubt with many as to the existence of such a condition, besides providing many jokes for the medical profession and laymen.

The suggestive symptoms and objective signs are many, so that the over-anxious surgeon or the would-be surgeon has no difficulty in blaming an innocent appendix for one or more of these symptoms. He advises its immediate removal and proceeds to remove it without further differential diagnosis, while the real cause of the trouble may be a peptic ulcer, a duodenal ulcer and stasis, ptosis, a stone in the right kidney or ureter or even a ptosis of this organ. When the pathologist renders a report of a fibrosis and makes a diagnosis of chronic appendix he feels fully satisfied that his diagnostic ability is wonderful and his surgery better. Six months to a year later the patient comes in for a diagnosis, saying that her symptoms were never relieved.

Our X-ray Department has condemned the use of the plain water and barium mixture in diagnosing this condition, and they question at times the correctness of observations made with this mixture. I wonder if Dr. Brown and his co-worker have found this as satisfactory as some food mixture where the barium is better held in suspension, and when the emptying time of the stomach is slower?

Surely no internist is justified in making a diagnosis of chronic appendicitis and no surgeon in removing a so-called "chronic appendix" until a careful differential diagnosis is made, regardless of the number of subjective symptoms and objective signs.

Dr. Gaither (closing).—I desire to emphasize a few of the points we have made in the paper. First, we do not wish to be understood as suggesting that this method insures a certain diagnosis of chronic appendicitis; it is a helpful, and not a final, means; for concomitant subjective symptoms and objective signs are of invaluable assistance in establishing a definite conclusion.

It may be of importance to state that we are fully cognizant of the fact that non-visualization of the appendix is no proof that disease of the appendix is absent.

It seems almost needless to add that in these cases, as in all we study, we endeavor to eliminate from the picture every other pathological lesion which may bring about the symptoms and signs presented before making a final diagnosis.

SICKLE CELL (MENISCOCYTIC) ANEMIA*

By GEORGE S. GRAHAM, M.D.,
and
SARAH H. MCCARTY, A.B.,
Birmingham, Ala.

Diseases of the blood hold a peculiar interest. No other of the body tissues offers its elements so readily to direct chemical and microscopical study, nor by the same token, does any other offer problems more baffling in their interpretation nor more interesting both for the practitioner and for the theorist. It is not surprising, therefore, that the announcement of a possibly new syndrome with outstanding hematological signs should be received with widespread interest. Such announcement came in 1923 with the paper upon "Sickle Cell Anemia," written by Sydenstricker, Mulherin and Houseal.[6] It was closely followed by Huck's report.[7] The original observation of Herrick[1] and the papers of Washburn,[2] Cook and Meyer[3] and Mason[4] had laid the foundation for a differentiation of the disease, while the observations of Emmel[5] had demonstrated more fully its characteristic erythrocytic anomaly. It remained, however, for Sydenstricker and Huck to develop the picture and to outline the problems presented. The disease or status has not yet been clearly defined

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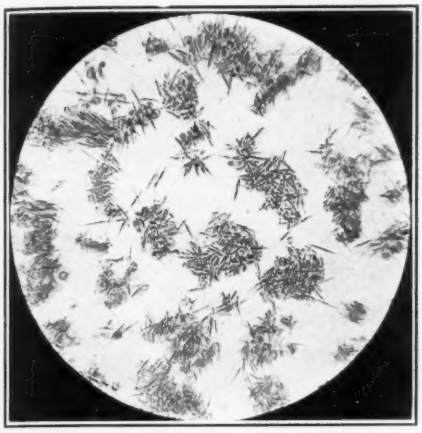

Fig. 1
Meniscocytic deformity of acicular type. Wet preparation.

either clinically or pathologically. Its real interest lies in the fact that it appears to have direct relationships with other blood dyscrasias and that it offers a ready source of material for the study of some, at least, of the factors concerned in the destruction of the red cells both within and without the body. We have nothing new to offer toward the solution of these problems, but wish merely to attempt a review of the more or less conflicting observations thus far recorded in the literature and to restate the case for the acceptance of the disease as a complex demanding further intensive study.

For the cytologist, the first arresting phenomenon is the startling deformity exhibited by the red cells when a drop of the fresh blood of an affected individual is placed on a slide beneath a coverglass and allowed to stand for several hours undisturbed. After an initial period of cytoplasmic unrest indicated by active kneading movements of the cell mass, the cells gradually, or at times suddenly, are transformed into peculiar crescent or semilunar or scimitar shaped bodies or into various angulated or burred forms while from the ends or surface angulations long cytoplasmic bristles may stretch outward for distances equalling or exceeding the cell length. In some individuals the tailed crescentic forms predominate. Again the cells are not so elongated, the projections are blunter and a more compact burred deformity results. We have encountered one individual in whom the cells drew out into long fusiform or acicular bodies with delicately tapering ends and high refractility (*Fig. 1*).

The deformity differs from any other type of poikilocytosis with which we are familiar. We have proposed for it the term meniscocytosis.[25] As used in the present discussion this term, with its corresponding adjective and substantive forms, is to be understood as referring merely to the presence in a given individual's erythrocytes of an inherent property for undergoing peculiar form changes when suitable conditions are provided. The anomaly is familial and is transmitted according to the Mendelian law as a dominant factor. Its frequency has proved to be much greater than was originally suspected. In a first series of 250 unselected negro patients, we found an incidence of 5.2 per cent. In a second summary[25] we had added 358 examinations and there were 7.2 per cent of positives for the entire series. We attributed the marked increase to the routine use of thin (No. 0) coverslips following the discovery that in an occasional specimen sickling might take place beneath such cover, but fail to appear when a thick (No. 2) coverglass had been used. Another element undoubtedly of large importance has been an increasing insistence on the use of scrupulously clean glassware. Intermittent returns to the routine setting up of preparations on unselected patients has given us a present total of 1,500 bloods examined. Of this total, 8.13 per cent were positive. Cooley and Lee[19] found 7.5 per cent positive, Miyamoto and Korb[31] 6.3 per cent, and Josephs[29] 6.4 per cent. The anomaly appears in both sexes and is usually accepted as transmissible by both. More recently it has been suggested that the mother is more directly responsible. Thus, of 15 mothers examined,[19] eleven were positive while only one of six fathers was positive. Of 300 patients examined by Miyamoto and Korb positive results were obtained in 4.8 per cent of the males, as against 7.1 per cent of the females. Our own results show a total incidence of 50 (8.7 per cent) among 556 males and of 72 (7.7 per cent) among 944 females.

Meniscocytosis, interesting as the phenomenon may be in itself, does not necessarily imply disease. Chauffard is quoted as remarking of familial hemolytic icterus that its victims were sometimes "more icterics than patients." Similarly, we have fallen into the habit of speaking of meniscocyte-bearers merely as "sicklers," leaving to further investigation the question as to whether or not they were sufferers from a peculiar anemia. Paraphrasing Chauffard's deli-

cate expression into the cruder English, it might be said that in many cases these people are more sicklers than sick. Sydenstricker[8] early recognized the difficulty and spoke of "active" and "latent" forms of the disease. He estimated roughly that active manifestations occurred in about 10 per cent of affected individuals. In the light of our own experience this estimate seems to be too high, but on the other hand, our percentage incidence of meniscocytosis is considerably higher than that originally reported by him. Much confusion has arisen from inattention to this distinction between meniscocytosis and meniscocytic anemia. Thus several authors conclude that the disease is of no clinical importance since they fail to find any obvious differences in the condition of sicklers and of non-sicklers in groups of negro patients. On the other hand Wollstein and Kreidel[41] report that in three autopsied cases they could find nothing but the anemia to account for death. Our own impression has been that the sickler who presents even mild anemia is a subnormal individual and even though he may not be regarded as an active case of sickle cell anemia, he is still ill equipped to withstand the vicissitudes of life and more especially the attacks of infection. Sydenstricker[8] has stressed the high childhood mortality in affected families and states that "a considerable proportion of the underdevelopment seen in negro children past the rickets age is due to this disease."

Our present knowledge of the disease has been developed in this country. The past six years have given us a rapidly growing list of reports from various parts of the United States. Little attention has been accorded in Europe although reviews of the American work have appeared in France[13] and in Germany.[28] A number of continental writers have, however, described crescentic erythrocytes in a varied list of diseases, particularly in malaria.[48] Bernhardt in Germany[24] and Castana[15] in Italy (both dealing presumably with white patients) have recently reported cases of anemia with crescentic erythrocytes in the blood smear. The latter identifies them definitely with the cells reported in sickle cell anemia and questions the existence of a separate complex such as has been claimed by writers in this country. Castana offers no pictures and his description is difficult of interpretation, but there is considerable doubt whether he or the other continental writers have dealt with the true meniscocyte. It must be admitted, however, that the question raised is a pertinent one and further reports will be awaited with considerable interest. Lawrence[30] similarly, has reported the occurrence of sickle cells in both healthy and ill white and colored individuals. Again, there is doubt whether he dealt actually with the cell under discussion.

The only undoubted case that we have found reported from abroad is that of Archibald.[17] The patient was a thirteen-year-old Arab boy of the Sudan. The case is of especial interest as bearing upon the question of racial specificity. There has been a widely adopted assumption that the disease is peculiar to or even specific to the full or part blood negro. This assumption has rested partly upon Sydenstricker's reported failure to find the anomaly in a systematic examination of 1,000 white patients,[8] and partly upon the similar failure of Emmel and subsequent workers to find it in preparations made from white patients who were suffering from various types of anemia. Stewart described a typical case in the child of a Cuban woman who denied negro ancestry.[33] More recently Cooley and Lee[47] report the occurrence in a Greek child as well as in its mother and brother. This case and that of Archibald emphasize the need for the investigation of this problem by observers abroad. The question has obvious ethnologic interest.

The clinical manifestations of the active disease have become too well known to deserve extended comment here. They center about the presence of a severe anemia of distinctly hemolytic type, with its attendant subjective and objective symptoms and signs. Fever is typical and the patients complain of rheumatoid pains in the extremities or joints and particularly of paroxysmal epigastric and hypochondriac pains with which may be associated nausea and vomiting. There is cardiac and renal disturbance and the liver is slightly enlarged. Edema of the ankles is frequent and in many of the active cases past childhood there have been ulcers of the legs or ankles. The subjects are apt to be underdeveloped physically and perhaps also mentally, or may show mental disturbance during exacerbation.[16] The sclerae have a characteristic greenish or greenish yellow coloration probably expressing an icterus that may sometimes be recognizable also in the skin or mucous membranes. The clinician who is not on his guard may easily be led astray in his diagnosis. In one of our cases the patient had been referred as a case of acute meningitis. In Stewart's case the early diagnosis was pernicious anemia. Other mistaken diagnoses include acute rheumatic fever, endocarditis, vertebral caries, and syphilitic ulcer of the leg. Hamilton's case [21] had been subjected to laparotomy because of violent abdominal pain uncontrolled by morphine. In two of our cases the diagnosis of acute appendicitis was followed by laparotomy which revealed a normal appendix in each instance. Smith[40] in particular has stressed the difficulties of differential diagnosis.

In the susceptible individual the period of onset of active symptoms is variable. A number of reported cases illustrate onset during infancy. [17 26 33 27 18] Again, the child may appear normal until it reaches the age of five to ten years. [3 9 14] There is a final period of onset during adolescence or early maturity as in Washburn's case (age twenty). Whatever its position

in the life curve, it is likely that the onset of active symptoms represents the point where an excessive rate of hemolysis is no longer compensated through some overactivity of the hematopoietic tissues or through other physiological check or balance. The shortening of life attributed to the disease by Huck is perhaps an expression of hematopoietic decompensation or exhaustion. Ganssle[11] notes a similar decompensation at about the twentieth year in his study of familial hemolytic icterus. It is probably significant that of 36 cases of sickle cell anemia sufficiently marked to warrant record by various authors, only three patients were over thirty years of age.[14 21 34]

The laboratory findings are decisive for the meniscocytosis. But as already noted, the presence of this anomaly is not incompatible with high red counts and hemoglobin values. In many of our cases the meniscocytosis would not have been suspected from the appearance of the stained smear or that of the fresh coverslip preparation. As we encounter individuals of the pre-anemic or anemic levels we find the well known signs of an active hemolytic disturbance. In the active case there may be surprising numbers of nucleated red cells. The reticulocytes may reach 30 or 40 per cent. The red count drops to 2,500,000 or even to 1,000,000, and the hemoglobin is depressed to a slightly greater relative degree. Oat shaped and crescentic erythrocytes are present in the circulating blood, sometimes in considerable numbers. The platelets are increased. The icteric index may reach figures of 18 to 21. The blood picture may change rapidly with fluctuations in the disease. Hamilton reports a count of 3,500,000 and a hemoglobin of 50 per cent, and five days later a count of 1,880,000, with hemoglobin of 38 per cent. During an extended period of observation with his patient in varying stages of exacerbation and remission, Smith noted variations in the hemoglobin between 65 and 35 per cent, and in the red count of from 3,665,000 to 2,055,000. The number of circulating sickles found in the blood appears to be an individual characteristic of the given patient rather than an index of the existing phase of the disease.[40] Significant urinary findings are a low fixed specific gravity, albuminuria and cylindruria, urobilinuria and decreased phenolsulphonephthalein output. The gastric acidity is reduced even to achlorhydria.

"Fragility" tests have given variable results. We should expect *a priori* to find a decreased resistance of the red cells. As a matter of fact, Huck found a slightly decreased, and Sydenstricker a slightly increased resistance. Normal values are reported by a number of writers.[33 22 41 20 17] Cooley and Lee find no difference as between 21 sicklers and twelve non-sicklers. They[19] report an average reading for the sicklers of 0.39 to 0.25 per cent NaCl, and for the non-sicklers of from 0.40 to 0.22 per cent NaCl. Hein, McCalla and Thorne find the downward range increased, and definite increase of resistance is reported by Mason and by Dolgopol and Stitt.[43] Smith[40] found a range of 0.50 to 0.20 per cent NaCl. The sickle cell of the circulating blood is said to be more resistant than the round form to the hemolysing action of hypotonic saline[46] and the same is said to be true of the reticulocyte.[23] The percentage of such forms present in the blood may influence the resistance readings. In a series of 24 meniscocytic bloods we found an average range of hemolysis of from 0.39 to 0.19 per cent NaCl. and concluded that the resistance was definitely increased. In a few instances, complete hemolysis occurred only in unbelievably low percentages of sodium chloride.

The peculiar deformities exhibited by the coverglass preparations are so unique that they excite immediate curiosity as to their causation. Emmel, who first demonstrated them, believed there was some abnormality both of the serum and of the cells. Huck concluded that the abnormality concerned only the cells. Sydenstricker found that the appearance of deformity was retarded by cold and accelerated by heat, as well as by bile pigment and by bile salts. Hein, McCalla and Thorne also found that sodium glycocholate hastens the change. Huck suspected some disturbance of surface tension and Josephs[29] found that cells that had been suspended in varying strengths of solutions capable of lowering surface tension exhibited the sickling deformity as well as agglutination in the tubes in which hemolysis was just beginning. Hahn and Gillespie found no relationship between the deformity and changes in osmotic pressure nor in changes in hydrogen ion concentration. Sydenstricker,[46] on the other hand, reports that a reduction in the hydrogen ion concentration to 7.3 or 7.25 causes immediate sickling of the cells *in vitro*.

There has been disagreement as to the behavior of the susceptible corpuscles when they are suspended in saline solution, when they have been received into oxalate solutions or when they are washed and replaced in serum or in plasma. Huck reported that the cells sickled in homologous normal serum but that serum from an affected blood would not produce sickling in homologous normal cells. The cells sickled normally in oxalated whole blood. Hein, McCalla and Thorne found that sickling does not occur in saline suspensions of affected red cells and Bell et al found no sickling in washed cells resuspended in their own or in foreign sera. Josephs washed affected cells repeatedly and found that when the cells were resuspended in the fluids from successive washings, sickling occurred in some of the fluids but not in others. He believed that sickling depended upon an adsorption by the cell of some plasma constituent and that this unknown substance could be washed off with saline.

Hahn and Gillespie[26] report that characteristic distortion of susceptible cells can be produced by subjecting them in saline suspension within a hanging drop preparation to atmospheres of CO_2, ethylene, nitrous oxide, nitrogen or hydrogen, and that immediate reversion to the round form occurs when these gases are replaced by O_2. They conclude that sickling is due to "progressive

decrease of oxygen and accumulation of CO_2 resulting from the continued metabolism of the blood cells." These findings are confirmed by Miyamoto and Korb, and by Landon and Lyman.[44] Sydenstricker in his last article[46] accepts them with reservations. We have failed repeatedly in our attempts to duplicate the experiment and have been unable thus far to find the fault in our procedure.

The theoretical implications of a direct relationship between meniscocytosis and oxygen deficiency are extremely interesting. Yet there are certain objections to the full acceptance of such relationship. We have reported[25] that in meniscocytic blood taken from the body after death the red cells are round (in some cases they may show a small percentage of deformed elements), but that after they have been mounted in the usual manner beneath a coverglass they undergo the meniscocytic change. This has been confirmed by Hein, McCalla and Thorne. If anoxemia can of itself produce the sickling deformity we should expect that the red cells would already be deformed in the vessels after death, or that at least no further change should take place in coverglass preparations in blood taken, as we have taken it, at intervals of as long as 27 hours after death.

In a recent experiment, we withdrew blood from the right auricle twelve hours after death into a flattened capillary pipette, containing a vacuum, and which was sealed off at both ends with the blow pipe before withdrawal from the heart wall. This blood contained a small percentage of sickle cells, estimated to be the same as had been observed in preparations made from the circulating blood during life. Observation over a period of one week revealed no change in the appearance of the red cells contained in the pipette. Coverglass preparations made at the same time showed typical sickling that was practically complete in one hour.

We have found that when fresh unsealed coverglass preparations of meniscocytic blood are placed in chambers filled with O_2 and CO_2 deformity proceeds in both at equal rates and to equal degree. A finding that appears to be directly opposed to that of Hahn and Gillespie is reported by Hein, McCalla and Thorne, who found that blood taken under paraffin oil and not exposed to the air shows no sickling deformity. Hahn's prediction that anoxemic patients would be found to exhibit a heightened rate of deformity and hemolysis is not borne out in the experience of Dolgopol and Stitt. The commonly observed presence of sickle cells in the blood smears and counting chamber would of itself argue against the reversal of sickle cells to the round form under the action of oxygen. This argument is weakened by Sydenstricker's report that the "circulating sickles" are not affected by oxygen in the gas chamber experiment. He regards these cells as different from the deformed cells that appear in coverglass preparations. It may become advisable to distinguish between the circulating sickle cell and the meniscocyte of artificial conditions.

Our own experience has led us to believe that the meniscocytic deformity is, at least largely, mechanical in its origin. That the red cells in affected individuals are noticeably soft and pliable was observed by Cook and Meyer, and by Emmel. When the blood of an active case is centrifuged the sedimented cell mass is thick and viscid ("stringy" is Joseph's term) and difficult of resuspension in saline. We early noticed a variability in the appearance of deformity in different areas of the coverglass preparation and in parallel preparations set up in apparently identical manner. Deformity was apt to appear first in those fields where the blood layer was thinnest and presumably under greatest pressure. Such areas were commonly in the middle of the coverglass area, but might be marginal. Oxygen deficiency should be relatively least marked in such fields since the cells are more widely spaced in the suspending fluid. It appeared also that the deformity could be obtained most certainly on glassware scrupulously clean and, in particular, free of oily material. Deformity was slight in hanging drop preparations, as noted by Emmel. When the coverglass is supported at the sides by coverglass fragments in such maner as to leave a central well, the cells of the thick layer in this well do not sickle but those compressed between the coverglass and the fragments sickle normally. Again there is no evident relationship to oxygen deficiency but rather the reverse.

The appearance of the meniscocytic deformity seems in our experience bound up with the clotting of the blood and more especially with the formation or perhaps, rather, the contraction of the fibrin net. Susceptible cells sickle slowly, if at all, when they are suspended in saline in high dilution, but when the saline dilution is slight, sickling occurs. Here, as well as in blood treated with anticoagulant salts, the sickling deformity appears first about the platelet clumps where often a fine fibrin net can be observed. Similarly, sickling is weak or uncertain when washed cells are replaced in their own or in homologous foreign serums, but pronounced when

they are added to uncoagulated plasma obtained without the use of anticoagulants. When susceptible cells are received directly into solutions of hirudin or of heparin, no sickling occurs in most cases, provided the technic is successful in the complete prevention of clotting. In about fifteen bloods thus treated there has been but one exception, that of an active case whose cells sickled even in the presence of these animal anticoagulants. In the other cases, the cells maintained the round form for one or two days, after which time some deformity might appear.

Supporting the mechanical concept of the deformity is the fact that light pressure upon the coverslip of the sealed preparation sufficient to displace the meniscocytes readily causes them to revert to the round form. There is no question of oxygen entrance, for the change can occur without destruction of the paraffin seal. Contrary to Levy's findings, too heavy pressure results in destruction of the whole preparation in the field pressed upon. Some of the resulting fragments remain attached to the coverglass, while others float in the fluid. We have suspected that the reversion to the round form reported by many writers as occurring in their preparations after periods of a few days to several weeks, was actually the result of disturbance of the fibrin net either through pressure from the examining lens or from the handling of the slide. We have not seen this spontaneous reversal in undisturbed preparations or not, at least, until decomposition changes have appeared. In line with this mechanical conception of the sickling deformity, we would call attention to the fact that in the hanging drop preparations of Hahn deformity is stated to occur in greater degree in cells lying at the edges rather than in the center of the hanging drop. Here is a suggestion that the changes may be due to surface forces, acting particularly upon the cells held within the surface film of the drop.

We have reported[25] that in parallel blocks of tissue fixed in Zenker's fluid and in formalin, distorted cells were found in the formalin fixed tissues, whereas the cells in the Zenker fixed material were round. Occasional deformity occurs, of course, particularly where shrinkage has allowed one-sided pressure upon the red cells, as immediately beneath the kidney capsule or at the free margin of a blood clot. In one case we found markedly elongated and trypanosome-like cells in the Zenker fixed spleen as reported also by Foote,[23] but in most instances the round form has been typical. Conversely, rounded forms may occur at times in the formalin fixed blocks, but deformity is typical. We regard the deformity in the formalin tissue as due to the irregular shrinkage permitted by poor fixation and hardening. Jaffe[28] does not agree with this observation but it is confirmed by Hahn and Gillespie who, however, explain the observed differences in terms of oxidation and reduction. We have found, however, in agreement with Hein, McCalla and Thorne, that fresh cells received directly into formalin are not deformed by any reducing action of the formalin even when placed beneath a coverslip in the usual manner. We have recently fixed corresponding blocks of tissue from the organs of a known "sickler" in different fixatives and embedded afterward in paraffin. The round or deformed state seemed to be a function of the degree of tissue shrinkage, rather than of the chemical nature of the fixing agent.

We have dwelt somewhat at length upon these varying observations and conclusions because they seem to bear upon the fundamental question as to how the meniscocyte differs from the normal cell in its structure and in its environmental relationships. The observations are admittedly crude in comparison with the delicacy of the forces and conditions that require measurement. Little is known of the physical chemistry of the red cell, and we have as yet little insight into the balanced forces governing the regeneration and destruction of the cell within the body or its destruction outside the body. The biochemist must give us information on this fundamental problem before we may hope to understand the phenomena expressed in the destruction of the red cell or even in its form changes.

Explanation of the hemolysis and anemia evident in this disease as well as in other members of a possible group represented by it must still be only a guarded speculation, with the chances, as usual, somewhat against the speculator. It is known that there is an unusually active phagocytosis of the red blood cells by the large mononuclears of the circulating blood, and by the fixed cells of the so-called reticulo-endothelial system. Erythrophagocytosis (if we may use a word to which some objection has been made) was observed in the circulating blood by Emmel and both Sydenstricker and Huck suggested that in it lay at least a partial explanation of the anemia. The large mononuclears of the affected individual do not take up normal red cells.[10] Sydenstricker suggested that the disease presents resemblances to familial hemolytic jaundice. This view has in substance been widely accepted.

There should possibly be added to this concept the hypothesis of the existence in the blood fluids of some substance inimical to the individual's own red cells. Sydenstricker reported the occurrence of auto-agglutination in four of his patients. We have ourselves failed to demonstrate agglutinins and others have failed similarly. [26 34] No autohemolysins have been demonstrated by the usual test tube method, but in agreement with Cooley and Lee, we have observed an increased rate of hemolysis when washed sterile cells were suspended in sterile physiological saline solution or in their own serum and placed in the incubator. Hemolysis is less evident when they are allowed to stand at room temperature, and absent at refrigerator temperature. We have also had ocular demonstration of relatively decreased stability of the red cell when suspended in its own fluids. Free hand and camera lucida drawings made from fresh coverslip preparations have demonstrated the spontaneous disappearance of erythrocytes in blood set up under sterile conditions. In such fresh blood of an active case we have watched an occasional or several cells per oil immersion field very gradually lose in color intensity and in the course of a few hours fade slowly to complete disappearance. There is no deformity, no change in size, no visible diffusion of hemoglobin, and no suggestion of the formation of sharply outlined ghosts such as are found in cells artificially laked. Whether the phenomenon depends on the presence in the blood of an autohemolysin in the usual sense of the term, or whether it evidences merely some inherent defect in the erythrocyte itself, we are unable to say. It may be that we see here in exaggerated form a process of erythrocyte destruction common to all blood. As far as concerns meniscocytic bloods, there are obvious differences in the destruction rate of red cells in different individuals. Such differences may be conceived as leading by insensible degrees back to the levels where meniscocytosis disappears and the blood becomes normal. We have observed bloods in which the peculiar form changes that precede meniscocytic deformity were active but in which no meniscocytosis could be demonstrated, and have suspected that such blood represented a transition stage between the normal and the abnormal as far as this particular character is concerned. It is not unlikely that in the observed susceptibility of the red cell to rapid and complete destruction within its own suspending medium we may glimpse the real crux of the whole problem and the point against which further investigation might well be directed.

Attention has already been called to certain points of resemblance between this disease and congenital hemolytic icterus. There is much in common. The main differences are the definite increase of "fragility" in the red cells of icterus and the usual tendency in that disease toward enlargement of the spleen. Both diseases appear to depend upon inherent constitutional defect. Gansslen has elaborated recently the hypothesis that there exists a "hemolytic constitution" that is inheritable, familial and transmissible as a dominant factor even through a mother who may herself show no evidences of the anomaly. Its bearers may exhibit any grade of abnormal intravitam hemolysis from absent or slight to the most profound. He hypothecates also an hematopoietic "compensation" that may be maintained with slight break or none, throughout a normal life span, but which may, again, fail at critical periods. We have theorized along this line for sickle cell anemia.[25] Gansslen points out that jaundice and anemia may be present at, or soon after, birth, or may make its appearance only during childhood or adolescence or at a frequent period of decompensation falling around the twentieth year. The same appears to be true of sickle cell anemia. Further, congenital hemolytic jaundice is accompanied frequently by such stigmata of constitutional inferiority as skeletal anomalies, underdevelopment or actual infantilism, and other vaguely defined disturbances such as have sometimes been attributed to endocrine dysfunction. Subjects of sickle cell anemia may also exhibit such signs. Both diseases are characterized by enlargement of the liver, and by attacks of paroxysmal abdominal pain of unexplained origin. The activating factor or factors back of the hemolytic crises are in both diseases difficult of discovery. Beitmann states that in hemolytic icterus exacerbation may be inaugurated by exercise, food, drink, excitement or exposure to cold, and Gansslen adds infectious diseases or endogenous causes. In neither case is there much, if any, of the usual evidences of biliary intoxication despite the high bilirubin content of the blood. Of interest in comparison with Huck's statement that sufferers from sickle cell anemia die by the thirtieth year of life, is Gansslen's report that the compilation of many family histories in hemolytic icterus has shown that individuals in whom the disease was active have died in the age period of thirty to forty years.

It can hardly be doubted that the spleen has some relation to the excessive blood destruction rate in sickle cell anemia, just as the same relation appears to hold in congenital hemolytic icterus. Yet in both cases there is surprisingly little histologic evidence of local blood destruction, except as it occurs in the former in areas representing old hemorrhage or infarct. While we have as yet no knowledge of what the splenic action may be, the well known curative effect of splenectomy in hemolytic icterus would encourage the hope that the procedure might be of equal value in sickle cell anemia. Thus far the results of the actual experiment have been none too hopeful.[37]

Sydenstricker first suggested the advisability of the operation and seven cases are now on record in which it has been performed.[26 33 23 19 35 44] All the patients have been children of eighteen months to nine years of age. In all but one case[33] the spleen was enlarged, reaching in one case[44] 621 grams in a four-year-old boy. Successive hospital admissions provided data in Stewart's patient from the age of sixteen months to that of six years, when operation was performed. The original examination revealed an enlarged spleen reaching 5.5 cm. below the costal margin. At two and one-half years it was 6 cm. below the costal margin, but at four years could not be palpated and when removed at six years it weighed only 46 grams.

The symptomatology in all the splenectomized cases has been typical of the active disease. The immediate results of operation are difficult of interpretation on account of the usual preliminary use of transfusion.[37] This procedure was valueless in Stewart's case, but it has usually appeared to exert marked stimulative effect.[36] This is suggested especially in the case of Landon and Lyman where an initial red count of 950,000 and hemoglobin of 20 per cent was increased to 3,600,000 and 44 per cent during an observation period of five months, during which time two blood transfusions had been given. For the most part, splenectomy appears to have been followed by a temporary rise in the red count and hemoglobin percentage and a prompt decrease in hemolysis as evidenced in the markedly lowered values for the serum bilirubin and for the urobilin of the urine and stool.[26] In Bell's case the reticulocytes increased in numbers continuously over a period of six months. Poikilocytosis, anisocytosis, polychromatophilia were always present, as were a few myelocytes. There was no change in the platelets. In Stewart's case there was no improvement in the patient's condition after splenectomy. Hahn reports early spectacular clinical improvement and Cooley and Lee consider their patient's hematopoietic system "stabilized" six months after operation. The patient longest under observation is that of Landon and Lyman. In this case the early effect of operation appears to have been satisfactory and the blood showed slowly rising red count and hemoglobin, persisting for some six months. Nine months after operation the patient was readmitted. The red count and hemoglobin were still at their previous higher levels, but despite this hematological improvement, there was an interval history typical of continued active sickle cell anemia. The liver had enlarged, the patient had gained no weight and was complaining of fever, paroxysmal abdominal pain, and pain in the ankles. Fourteen months after operation the red cells stood exactly at the pre-operative (but post-transfusion) level and the hemoglobin was but a few points higher. In Bell's case the improvement in the blood picture noted five weeks after operation was less marked at two months and at six months the red count was 1,240,000 as contrasted with 2,000,000 before the operation. The hemoglobin of 50 per cent was slightly above the pre-operative level of 40 per cent. The early improvement in the icteric index has also proved to be temporary, at least in some cases.[35 23 44] Operation has had varying effect upon the resistance of the red cells. No change in the resistance was found in one case.[19] There was a marked decrease in the resistance in the case of Hahn and Gillespie. In Bell's case there was an extraordinary shift in the opposite direction. Here a pre-operative resistance curve of 0.3 to 0.2 per cent NaCl was found two weeks after operation to have changed to one of 0.28 to 0.16 per cent NaCl and at six weeks hemolysis began at 0.30 per cent and was not complete even in distilled water. The same was true six months after operation. A similar gradual increase in resistance is evident in the case of Landon and Lyman. As might be expected splenectomy has no effect on the meniscocytosis. It has been suggested[35 46] that the spleen is enlarged during the early stages of the active disease but undergoes atrophy as time goes on and that splenectomy may offer its best results in the cases with splenic enlargement. But in the quoted case of Landon and Lyman the spleen was unusually large and splenectomy appears distinctly to have failed except temporarily to arrest the course of the disease.

We have had opportunity for the post mortem study of eleven patients with meniscocytosis. Two of them were recognized only at autopsy. We have been unable to review the autopsy material of the past years, but there is no doubt that we have failed to recognize many cases, and also no doubt that we could pick out at least some of the positive cases by study of the sections. Differentiation could be accomplished easily in formalin fixed material on the basis of the red cell deformity alone, for this is highly characteristic. In only one of our cases (and that was the first studied) were the lesions so unusual as to have appeared particularly interesting to the uninitiated. We should have been quite at a loss to explain the gross and microscopic changes in that case had we not already studied the patient in the ward in the light of the then recent publications of Sydenstricker and Huck. Bennett[42] found a similar case equally puzzling. Here the spleen of a twenty-year-old patient weighed only 10 gm. Death had occurred in the engorgement stage of a bilateral lobar pneumonia. In many cases the histologic changes are so obscure as almost to escape notice. Cooley and Lee, after studying three cases at autopsy, conclude that there is no lesion peculiar to the disease. In our own series we have encountered cases with barely recognizable stig-

mata and others with varying degrees of histologic change to the most extreme.

The commonest stigma of the disease is the evidence of a long continued hemolysis. Phagocytic endothelial leucocytes with engulfed red cells or blood pigment may be encountered in the vessels of the various organs, but particularly in the liver sinusoids and in the lymph nodes, less commonly in the spleen and bone marrow. The Kupffer cells of the liver are usually actively phagocytic and may be stuffed with red cells or with iron-containing pigment. This was true in the case of a twenty-nine year old man, whose blood had contained 95 per cent of hemoglobin and 4,160,000 red cells per cu. mm. and who died of an extra-dural hemorrhage three days after he had suffered cranial trauma. Even in the slightly developed case, at least in individuals below forty years of age, there is little or much iron-containing pigment in the epithelium of the Henle's loop, especially in the ascending limb. Occasionally it appears also in the epithelium of the convoluted tubules or in the intermediate segment. In a two-year-old child with moderate anemia there was abundant pigment in the convoluted tubules but very little in the loop of Henle. Such distribution has not been usual in our experience, the pigment tending to appear first in the loop epithelium. As in hemolytic icterus, blood pigment deposit in the splenic parenchyma is little in evidence. There may be some pigment deposit in the lymph nodes and in the bone marrow. In the younger patients there is slight to marked hyperplasia of the bone marrow with evidence of overactivity both in the red and in the white cell formation. The megakaryocytes may be increased. There is sometimes a fine reticular sclerosis. In our first case, which has continued to be for us the prototype of the highly developed disease picture, sclerosing changes were very evident.

The most characteristic lesion is found in the spleen. Here there is reticular fibrosis and a marked engorgement which may or may not distend the sinus spaces but uniformly stuffs the pulp cords with red cells. So distended are the pulp columns that the proper splenic cells appear to have been displaced and the parenchyma "looks like a blood clot."[23] While splenic engorgement is found also in pernicious anemia and particularly in congenital hemolytic icterus, Rich[39] states that in the present disease there is a pathognomonic lesion consisting in an anomaly of the sinuses in the marginal zone about the Malpighian bodies, that leads to the formation of pools of blood immediately about these structures. This is referred to congenital imperfections of the ampullae of Thoma. There may also be an abnormal development of capillaries throughout the bodies. As far as we know, Rich has not yet published a detailed description of the lesion and it would be premature to attempt comparison with our own observations. We can, however, confirm the frequency of an engorgement of the marginal zone. It has been absent in only two of our cases, and both were in the older age group (forty and forty-six years). In one case there are distinct varix-like capillary distentions about the corpuscles. Subendothelial hyaline deposit may appear in the arterioles of very young patients. As time goes on the Malpighian bodies appear to diminish in size, sometimes very markedly. In one of our cases there is a sheath-like fibrosis about the body and in our first case the bodies are small and scarred, the central artery often embedded in a fibrous mass. Infarction had occurred in three of our cases, one a baby of four months, another a young man aged nineteen years, while the fully developed picture of extensive old hemorrhage or infarction with resulting scarring and extensive iron-containing pigment deposit was present in our first case.[19] Generalized fibrosis together with the occurrence of such grosser scarring results in the atrophy typical of the end stages of the splenic lesion. It is probably true that the spleen is originally enlarged, but this is not invariable since in the four-months-old infant with otherwise typical findings of generalized fibrosis and pericorpuscular engorgement the spleen weighed only eleven grams. In our first case the spleen in an adult weighed 28 gms., it weighed 10 gms. in both Jaffe's cases (aged eight and ten years), it was small also in Huck's case and in Sydenstricker's active cases. It was "shrivelled and small" in the twenty-year-old subject of Hein, McCalla and Thorne. Yet in other apparently typical and well developed cases, it has been of normal or above normal weight.[41] [32]

The urinary findings point to interference with the kidney function, but there is little to be seen microscopically beyond the pigment deposit. Our original suspicion that there might be a common occurrence of glomerular nephritis has not been borne out by further observation. The glomeruli usually appear swollen so that they fill the glomerular space, and the capillary loops are injected. Occasionally there are a few red cells within the space. But that is all.

The liver is typically enlarged and there may be bile stasis, but little or no blood pigment is found. There may be a fine intralobular fibrosis. Here as in other extramedullary locations a few or many marrow elements may appear in the active cases. In organs other than these mentioned there is nothing characteristic. Small wonder that the study of inactive cases has been disappointing. Only through an understanding of the variations in the findings can we reconcile the histology to the concept of a definite disease.

In our eleven autopsied cases, only one can be considered as a definitely established instance of active sickle cell anemia. This man, aged thirty years, died of acute streptococcic bronchopneumonia. Three other patients died of acute bronchopneumonia (aged four months, two years and nineteen years). Cultures from two of these yielded a streptococcus. In a boy of nine years death occurred three days after compound fracture of the femur and a hemolytic streptococcus was cultured from spleen and liver. A man twenty-three years old died of tuberculosis affecting chiefly peritoneum and pleura: a woman thirty-five years old died of typhoid fever, and a man aged twenty-nine years of cranial injury. Of the patients aged forty years or more, one (about forty years old) died of self-imposed starvation (psychotic), a forty-six year old man of chronic nephritis and a woman of fifty-seven died under anesthesia during removal of a carcinoma of the breast.

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INDICATIONS FOR LAMINECTOMY IN SPINAL INJURIES*

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In the chapter on spinal injuries in a recently published "System" on the "Practice of Surgery," I find the following statement:

"With a paraplegia total, both sensory and motor, operation is probably useless, but if the man or the family fully understanding this, requests it, it is to be done as a last chance. If, on the other hand, there is any motor or sensory function remaining below the dead line to show that the cord is not destroyed *in toto* the operation is to be considered and should usually be done."[1]

In general, the above statement may be true. In particular, to follow it is to employ a "hit or miss" method in arriving at the important decision as to whether or not a laminectomy is indicated. No matter how good a neurologist the surgeon may be, he is only able to guess that this or that traumatic lesion of the spinal cord is or is not a so-called complete or partial one.

Many years ago Bastian laid down the principle that in a complete transverse lesion of the spinal cord there is an abolition of all superficial and deep reflexes below the level of the lesion. That this is not always the case has been shown by the careful observation of complete

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spinal cord lesions during the recent World War.[2 3] This point is emphasized merely to prove that there are no hard and fast rules which will permit one, by a neurological examination alone, to say definitely that there is a complete transverse lesion, and operation is therefore contraindicated.

The object of laminectomy in spinal injuries is to remove pressure on the spinal cord, thereby giving the cord a chance again to transmit impulses which may have been interrupted by the compressing agent. If the block is simply a physiological one and not anatomical there may occur a return of function. If, on the other hand, there has occurred an actual division of the pathways within the cord, there will be no return of function even though all pressure on the cord be removed. One can not say definitely that a cord has been hopelessly injured until it has been actually seen, and even when actually inspected the cord may appear almost normal, yet a hopeless transverse lesion may be present.

To follow the advice quoted above would mean on the one hand that many useless laminectomies would be done, and on the other hand that many cases would be treated conservatively which should be operated upon. Merely that an individual case may show evidence of a partial lesion, is no reason in itself for operation. Unless the partial lesion be caused by something pressing upon the cord there would be no object in operating, provided of course that it is agreed that the object of the operation is to remove pressure.

Pressure on the cord as the result of injury may be due to several factors. Perhaps the most frequent of these is the displacement of bone fragments. Pressure may also be exerted by a narrowing of the spinal canal at the site of displacement. Collections of blood, extradural or intradural in location, may cause sufficient pressure to produce a physiological interruption of impulses in the cord. Edema of the cord *per se* with swelling is generally recognized as a cause of compression. Allen[4] in a series of experiments in which he produced an edema of the cord, found that permanent damage of the cord elements resulted within a few hours after the edema was produced. Late cord compression may result from organized exudates.

Since the jugular compression test was first described by Queckenstedt[5] we have a very simple and reliable method of determining whether or not the spinal canal is obstructed. Queckenstedt first employed the test as an aid in diagnosing spinal cord tumor. He found that the spinal fluid pressure could be increased by compressing both jugular veins. The explanation is simple. The brain is enclosed within a solid bony encasement. If the space be encroached upon by an overdistention of the veins and venous sinuses within the cranial cavity, the intracranial pressure will be increased. This can readily be demonstrated by doing a lumbar puncture and measuring the spinal pressure with either a mercury or a simple glass tube manometer. The jugular veins are now compressed. This causes a venous congestion of the brain which in turn forces the cerebrospinal fluid down the spinal canal. The manometer will immediately record the rising pressure. As soon as the compression of the jugulars is withdrawn the pressure falls to the original level. Queckenstedt demonstrated that a spinal cord tumor of sufficient size to encroach upon the spinal canal would cause either a partial or a total obstruction to the fluids being forced below the level of the tumor. Under such circumstances jugular compression would fail to influence the pressure below the tumor if the canal were completely obstructed. If the obstruction were partial there would occur a gradual and step-like rise in the pressure below the tumor, and a slow step-like fall on the release of jugular compression.

Coleman[6 7] first used the Queckenstedt test in cases of spinal injury in order to determine whether or not the spinal cord was sufficiently pressed upon to cause an actual obstruction to the spinal canal. If the test showed an obstruction, Coleman considered this to be a positive indication for laminectomy. On the other hand, as long as the canal remained open the test would show no obstruction and laminectomy therefore would not be indicated.

The jugular compression test, therefore, gives us a simple and reliable method of determining whether or not the spinal cord is being pressed upon as the result of a spinal injury. If it is pressed upon, a laminectomy should be done immediately, regardless of the neurological findings. The cord may be found hopelessly damaged and the operation therefore may be of no use as far as recovery from the paralysis is concerned. Such a condition, however, could only be guessed at by neurological examination alone. On the other hand, the cord may be found intact and affected only secondarily through pressure. The removal of the pressure may permit a complete restoration of function, even though the neurological

picture may have been that of a complete transverse lesion.

Three cases have been selected from a large series to illustrate the value of the test in causing one to decide for or against operation.

Case I.—A fracture-dislocation of the first lumbar vertebra with evidence of partial motor and sensory paralysis. No complete block on jugular compression test. No laminectomy. Recovery.

H. W. E., a white man, twenty years of age, was admitted to the Piedmont Hospital on January 14, 1927, one hour after he had fallen for a distance of 55 feet when the scaffolding broke on which he was standing. He was unable to move either leg immediately after the accident. The roentgen ray examination disclosed a fracture-dislocation of the first lumbar vertebra. The neurological examination revealed an inability to move either leg, the paralysis being of the flaccid or lower motor neuron type. There was an incomplete sensory paralysis below the first lumbar segment, in that touch and pain sense were only obtunded over the legs and feet, although there was an area of complete anesthesia around the anus and over the scrotum and penis. There was loss of bladder and rectal sphincter control. The patellar reflex was sluggish on the right side and absent on the left side. The cremasteric and plantar reflexes were absent.

The spinal fluid was under 110 mm. (water) pressure and was slightly bloody. On compressing the jugular veins the pressure rose in a step-like manner to 250 mm. On releasing the compression the pressure gradually descended to 160 mm.

The impression gained from the examination was that there was a partial paralysis but that the canal was only partially obstructed. A laminectomy therefore did not seem to be indicated at the time.

The following day there was a marked improvement in that the patient could partially move the legs. The jugular compression test was repeated, and there was no evidence of a complete block. The improvement in the paralysis continued. Ten days after the accident the compression test gave no evidence of pressure on the cord. The sphincter paralysis persisted. One week after the accident a suprapubic drainage tube was inserted in the bladder by Dr. M. L. Boyd. When the patient was discharged on April 4, 1927, there had occurred a complete return of sensory and motor function with the exception of some weakness of the ankles and toes. With properly fitted shoes he was able to take steps alone. The patient eventually made a complete recovery.

Case II.—Fracture-dislocation of the first and second lumbar vertebrae with evidence of complete motor paralysis and partial sensory paralysis. Complete block on jugular compression test. Laminectomy. Recovery.

M. A., a white man, 21 years of age, was admitted to the Piedmont Hospital on January 28, 1926. On January 22, 1926, one week before admission, while in a sitting position a log had fallen on him, crushing him forward. His legs became paralyzed immediately (both motor and sensory). Since the accident until the time of admission to the hospital he had been unable to move the legs or pass his urine voluntarily.

On examination there was a loss of pain sensations over both legs and over the lower portions of the abdomen up to a level 4 cm. below the umbilicus. The sensation to touch was present but obtunded over a similar area. There was a complete loss of voluntary movements of both legs and feet. The patient had to be catheterized and the urine contained large quantities of pus. The cremasteric, tendo-Achilles, patellar and plantar reflexes were absent. The roentgen ray examination disclosed a fracture of the first and second lumbar vertebrae with a dislocation to the right.

On lumbar puncture the spinal fluid was yellow and was under 140 mm. (water) pressure. On compressing the jugular veins there was no increase in the pressure.

The impression gained from the examination was that there was a definite compression of the spinal cord (conus and upper portion of the cauda equina) causing a paralysis of the sphincters and of the lower extremities. The fact that the sensation to touch was present although obtunded seemed to indicate a physiological rather than an anatomical block. In spite of the lapse of one week since the accident a laminectomy seemed indicated.

Upon operation, January 30, 1926, the spines and posterior arches of the twelfth dorsal and first and second lumbar vertebrae were removed. The first lumbar vertebra was not only dislocated toward the right but was rotated on its long axis. Within the spinal canal there was a large extradural organized blood clot. This was removed. The dura was then split longitudinally to the extent of the exposure. The conus and upper portion of the cauda equina were under such marked pressure that they actually protruded through the opened dura. In order to make sure that the compression had been relieved, the jugular veins were compressed by an assistant. This caused a free escape of spinal fluid from above downward. The dura was left open and the remaining wound closed in layers.

The day following the operation the patient was able to move the legs slightly. The improvement was progressive and the patient walked out of the hospital without the aid of crutches five weeks later. While in the hospital a suprapubic tube was placed in the bladder by Dr. M. L. Boyd. The patient still wore this tube on discharge. A letter from his physician, Dr. W. C. Morrow, of Andrews, South Carolina, on July 17, 1926, stated that the bladder and rectal sphincter control had practically returned to normal and that the patient was walking with the aid of a cane.

The importance of repeating the jugular compression test daily when no block is first demonstrated is illustrated by the following clinical example:

Case III.—Fracture of the second lumbar vertebra without dislocation. Immediate paralysis of the right leg. No complete block on jugular compression test on admission to the hospital. Evidence of partial block three days later, and evidence of complete block on the fourth day. Laminectomy. Recovery.

W. H. M., a white male, 19 years of age, was admitted to the Grady Hospital on October 18, 1928. Shortly before admission the patient had fallen from a scaffold for a distance of thirty feet. He was rendered unconscious for a few minutes. When he regained consciousness he could not move the right leg. The patient was admitted on the general surgical service and was not referred to the neurological service until October 21, 1928. At this time he complained of pain radiating down both legs, as far as the foot on the right side and as far as the knee on the left side. He was able to

move the left leg and foot normally. He was able to move the right foot and flex the right knee to a limited extent. There was an absence of cremasteric and patellar reflexes. The sensation to touch seemed normal over the left leg, and present though obtunded over the right leg. There was a loss of joint sense of the toes of the right foot. The roentgen ray examination revealed a fracture of the second lumbar vertebra.

A lumbar puncture done on admission (October 18, 1928) gave clear spinal fluid and there was a prompt rise in pressure on compressing the jugular veins. On October 21, 1928, the jugular compression test was repeated. At this time the fluid was slightly yellowish and rose slowly in the tube manometer when the jugular veins were compressed. On release of the jugular compression there was a gradual fall to a level somewhat higher than the original one. This was interpreted as indicating a partial block of the spinal canal. On October 22, 1928, the test was again repeated; the fluid was quite yellow in color and there was evidence of a complete block on jugular compression.

The impression gained from these examinations was that there was no block of the spinal canal on admission and therefore no indication for laminectomy. Four days later a definite spinal compression had developed, which indicated the necessity of performing a laminectomy.

October 22, 1928, operation was performed by my associate, Dr. Hugh Cochran. The spinus process and the posterior arch of the second lumbar vertebra were removed. The lamina was found to have been fractured and a fragment of bone was pressing inward against the dura. This was removed. The dura was opened and the underlying cauda equina seemed normal. The dura was left open and the remaining wound was closed in layers.

The day following the operation the severe pains in the legs had disappeared. Two days later there was a return of the appreciation of the position of his toes in the right foot and the cremasteric reflex had reappeared on the right side. From this time on the patient continued to improve and a few weeks later was normal in all respects. An examination in October, 1929, failed to reveal any abnormal findings.

Many more examples could be given to illustrate the importance of the presence or absence of a spinal block as a determining factor in deciding for or against laminectomy. It is felt, however, that the three cases already cited are sufficient. No reference has been made to those cases of spinal fracture without evidence of spinal compression or injury. Such cases are orthopedic and not neurosurgical problems, and are not within the scope of this paper.

Should one depend upon the evidence of spinal block as an indication for laminectomy he will very naturally operate upon many complete anatomical lesions which will show no subsequent improvement. On the other hand, he will operate upon many who will show little or no improvement if treated conservatively, yet who show marked improvement when the pressure is relieved. Furthermore, he will not operate upon many in which an operation would be a useless procedure, when there is no compression to remove.

Laminectomies for traumatic compression of the spinal cord can be done most satisfactorily under local infiltration anesthesia, and can thus be made a procedure with little or no shock. Under such an anesthesia, however, manipulation of the cord should be carefully avoided, as otherwise great surgical shock will be produced. No laminectomy in traumatic cases is complete unless the dura is opened freely and left open.

In conclusion I wish to emphasize the following:

(1) The degree of cord damage in spinal injuries can not be estimated accurately by a neurological examination alone.

(2) The roentgen ray evidence of dislocation is not in itself proof of spinal compression.

(3) The jugular compression test as described by Queckenstedt is the most reliable method of determining the presence or absence of pressure on the cord.

(4) Laminectomy should be performed only in those cases of spinal injury in which pressure on the spinal cord has been thus demonstrated.

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DISCUSSION (*Abstract*)

Dr. Isidore Cohn, New Orleans, La.—Dr. Dowman's paper is a plea for surgeons to make use of the valuable Queckenstedt phenomenon.

Too many patients have been operated upon for vertebral injuries.

Apparently none of Dr. Dowman's cases was in a condition of shock. One might gain the impression that some of these patients may be operated upon immediately. If we operate immediately, particularly in shock, we will get into trouble. Spinal puncture relieves pressure from the cord. After the patient recovers from shock, we can proceed with operative measures.

Many of the fractures do not need operation. I call to mind two instances within the last six months where the individuals had been in automobile wrecks. One of the patients had a fracture of the tenth dorsal and

the other a fracture of the first lumbar. Both of these patients walked five and seven days, respectively, without any medical attention. There was a slight rise in the intra-spinal pressure; when this was relieved plaster casts were applied. At the end of six months those individuals discarded their jackets and they are walking around comfortably.

Dr. Guy A. Caldwell, Shreveport, La.—If I have gained one lesson more than another from Dr. Dowman's talk, it is the importance of the repeated Queckenstedt tests. After the first negative test in cases of spinal injury, if the symptoms are progressive, the test should be repeated on subsequent days. When there is evidence that the spinal canal is blocked, then and only then should we do a laminectomy.

Dr. Frank P. Strickler, Louisville, Ky.—The lumbar region offers the best prognosis for fractures of the vertebrae. In this region the canal is larger and the cord has divided into the cauda equina, making it much more difficult to compress the cord.

I should like to ask Dr. Dowman what his results have been with fractures in the cervical and dorsal regions? He has excellent results for the lumbar region.

Dr. Dowman (closing).—In regard to Dr. Strickler's question, it is a well known fact that the higher the injury the worse the prognosis. In the dorsal region the canal is not very voluminous. Where there occurs a dislocation and reduction almost simultaneously it is easy for the cord to be crushed when the injury is in the dorsal region. Dr. Strickler has brought out an excellent point, namely, that in fractures of the first and second lumbar vertebrae the prognosis is better than when the lesion is higher. Cervical injuries, particularly where there is evidence of cord damage, are very grave problems. Many of these cases suffer from extreme shock and develop a marked hyperpyrexia. I often hesitate to operate upon cervical fractures with evidence of cord damage, even in the presence of a block, because of the extreme shock and the fact that practically all these cases die regardless of the type of treatment employed.

Dr. Caldwell has emphasized a point brought out in one of the case reports, namely, the importance of repeating the jugular compression test. As the third case gave no block at first, a partial block a few days later, and a complete block on the day following this, the value of repeating the test is thus proved.

In performing the Queckenstedt test a few points should be kept in mind. In the first place a simple glass tube manometer, about 0.5 cm. in diameter connected by a piece of rubber tubing to an adapter which fits the lumbar puncture needle, is all the apparatus which one needs. In the second place the patient should be placed on the side and enough novocaine solution injected to produce a perfect local anesthesia. By avoiding the production of pain in performing the puncture a very valuable diagnostic method will not be brought into ill repute. In the third place, the patient should be cautioned not to strain nor to cough as otherwise an overdistention of the spinal veins will be produced which will cause the fluid within the tube to rise. When the patient is perfectly quiet and gentle pressure is made over the jugular veins there occurs an immediate rise of the fluid within the tube provided there is no block present. If a partial block be present there will occur a gradual and step-like rise of the fluid when the jugulars are compressed and a slow step-like fall when the compression is released. In the presence of a complete block, jugular compression will cause no rise.

In the presence of a complete block laminectomy is indicated. Where the canal is open or only partially obstructed I do not consider laminectomy necessary.

OSTEOCHONDRITIS DISSECANS*

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and
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Several cases of this condition occurring in our clinic have suggested a review of the literature. Most of the authors have considered osteochondritis dissecans as of rather infrequent occurrence, but this has not been our experience. We believe that it occurs not infrequently, but is perhaps not recognized, or perhaps is diagnosed incorrectly.

According to Geyman, Konig, in 1888, first described this condition as a segregation of a portion of cartilaginous and subchondral tissue into the joint, applying to it the term "osteochondritis dissecans." Since that time several writers have contributed to the knowledge on this subject. Freiberg and Woolley, in 1910, cited a case of a girl who had had loose bodies in the knee removed while she was in Russia. The symptoms recurred and they operated upon her. They found a body attached by a pedicle with no depression of the cartilage or bone. Ridlon, in 1913, presented three cases. Freiberg, in 1923, presented five cases. Axhausen, in 1924, contributed a complete resume up to that date, discussing the pathogenesis in great detail. Schirmer, in 1925, presented four cases, while Balensweig added one in the same year. McCammon, in 1927, added one, and in 1928 Geyman added several more. Buchman has contributed several articles, three dealing with the spine, and one in 1929 covering all phases of the osteochondritides. Several others have contributed cases and have discussed the etiology.

The pathology of this condition is a localized aseptic necrosis of cartilage and the underlying bone, described as occurring typically on the articular surface of the inner condyle of the femur. This usually occurs in young adults.

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The location, however, is subject to considerable variation as will be pointed out later. The gross appearance on operative exposure is that of yellowish, apparently necrotic cartilage, which is not firmly attached at the base. When this cartilage is removed, a piece of bone usually accompanies it, leaving a cavity composed of rather soft, spongy bone. Histologic examination shows hyalinized fibrous tissue, and evidence of inflammatory reaction.

The pathogenesis of osteochondritis dissecans has not been definitely determined. In general, opinion is divided into two groups, one holding that it is of traumatic origin, the other that it is embolic in origin. Konig suggested that in some cases occasional trauma caused a crushing of the nutritive blood vessels, followed by a secondary necrosis. In other cases he believed that there was a non-traumatic origin, not determined. According to Schirmer, Borsinger roentgenologically examined injected knee joint specimens. These showed that certain branches of the genu media artery in childhood always represent terminal arteries, and may remain as such during life. Thus a blockage of these vessels by emboli might cause an aseptic necrosis such as is found in osteochondritis dissecans.

The clinical picture of these cases varies with the location of the process. Weakness and discomfort in the joint involved are the usual outstanding symptoms. After separation of the small bone button, locking of the joint may occur, with symptoms corresponding to the degree of interference with function. Many of the cases give a history of trauma, but its importance has been discounted somewhat, on the same basis as the frequent traumatic history of carcinoma of the breast cases as given by the patient.

The diagnosis can be made solely on the roentgen findings which are quite typical. There is an area of rarefaction of varying size, with smooth borders, in the subchondral region of the involved joint. In this area of rarefaction, there may, or may not be a bone button. In late cases, the bone fragment has become completely detached from its base and is a loose body in the joint. In the latter instance, the area of rarefaction is usually definitely demonstrable.

The usual treatment is removal of the diseased cartilage and attached bone button, whether *in situ*, or as a loose body. Complete restoration of joint function usually follows.

We are briefly presenting several cases occurring in our clinic, some of which occurred in an unusual location.

Case 1.—A white woman, twenty-four years old, was admitted December 10, 1921, complaining of pain in her left knee. Seven weeks before her admission, the patient was seized with a sudden pain in her knee, which continued up to the time of admission. Physical examination was negative, except for pain between the condyles of the femur on marked flexion. There was some tenderness in the same region. Roentgen examination was negative at this time. February 6, 1922, the patient returned with clinical signs of a pathological condition of the knee. On February 27, roentgen examination showed an irregularity of the texture of the patella. Operation on March 6, 1922, gave negative findings except for synovitis and a softened area of cartilage and bone about 1.5 cm. in diameter on the under surface of the patella. This was curetted away. The patient is now entirely well. At the time of examination and operation, the true nature of the condition was not known, but in view of our later findings, we feel that this is a definite case of osteochondritis dissecans.

Case 2.—A colored man, forty-three years old, was admitted April 16, 1926, complaining of pain in the right knee following a fall six weeks before admission. On examination the knee was slightly swollen, flexed, and was tender to deep palpation. Roentgen examination showed a marked deepening of the intercondylar notch, with local rarefaction of the bone just proximal to it. The diagnosis was "probable atypical osteochondritis dissecans." Operation revealed a yellowish lipomatous mass hanging from the location noted in the roentgen examination. The cartilage was rather friable. This case is considered as a very atypical case of osteochondritis dissecans, and the diagnosis is possibly open to question.

Case 3.—A colored woman, twenty-two years old, was admitted November 12, 1926, to the obstetrical service. While she was in the hospital a history was obtained of a "rheumatism" in her knee of seven years' duration. The knee was examined roentgenologically, and atypical osteochondritis dissecans was demonstrated, with the button acting as a loose body in the joint, and with an area of rarefaction on the outer condyle of the femur. The loose body was removed surgically, and was found to correspond exactly to an area of erosion of bone and cartilage previously demonstrated by the roentgen ray.

Case 4.—A white boy, sixteen years old, was admitted June 5, 1928, complaining of weakness of the left knee dating from childhood. There was a history of several injuries to the knee. At the time of admission, the patient used a cane. On examination there was an extreme degree of weakness, but no limitation of motion. Roentgen examination showed an atypical area of rarefaction on the articular surface of the internal condyle of the femur, with a small bone button 2 cm. in diameter lying in the center of this area. It was removed surgically.

Case 5.—A white boy, seventeen years old, was admitted June 8, 1928, complaining of weakness and loss of weight. He had limped for two years. Excessive walking caused weakness and pains in the hips. Physical examination elicited a click in both hip joints on rotation. Roentgen examination demonstrated typical areas of rarefaction with bone buttons *in situ*, in the heads

of both femurs. The patient's symptoms were not severe enough to warrant removal, so he was discharged without operation.

Case 6.—A white boy, seventeen years old, was admitted September 17, 1928, complaining of weakness and discomfort in both knees, of three or four years' duration. Roentgen examination showed an atypical area of rarefaction with a bone button on the internal condyle of the right femur. The lower end of the left patella showed a similar area of rarefaction. Both knees were explored surgically. On the left, the cartilage over the patella was somewhat thinned and presented a yellowish glistening appearance. All loose cartilage was dissected away. The right knee showed necrotic cartilage, but this could not be removed, as it was thought unwise to prolong the anesthesia. Seven months later the patient came back for a check up, and clinically was almost entirely relieved. Roentgen examination showed an almost normal left patella. The bone button and area of rarefaction were still present in the right knee.

Case 7.—A white boy, ten years old, was admitted May 20, 1929, complaining of a knee flexed at a forty-five degree angle, which could not be straightened. The patient woke up one morning six weeks prior to admission with this flexion of a previously normal knee. The deformity had continued without change up to the time of admission. Roentgen examination showed an area of rarefaction with a shell-like small fragment of bone lying just distal to the internal condyle of the left femur. At operation, the button could not be found, and it had evidently moved from its former location. The patient regained perfect function, and the joint has not locked again.

Case 8.—A white man, twenty-three years old, was admitted August 22, 1929. He had complained for some time of "knee trouble," that is, weakness and pain on motion. Physical examination showed tenderness over the external condyle, with fluid in the joint. Roentgen examination showed a large fragmented area of bone 3 cm. long, lying in an area of necrosis on the articular surface of the external condyle of the femur. This patient has not yet been treated surgically.

Case 9.—A white woman, thirty-two years old, was seen in the out-patient department September 23, 1929, complaining of recurrent slipping of both patellae. Physical examination showed a roughness or grating of the patella in both knees, and pain under the patella on forced flexion. Roentgen examination showed an irregularity of structure of both patellae with an atypical bone button in one.

The next three cases are quite doubtful, and are brought in merely to provoke discussion.

Case 10.—A white boy, fourteen years old, was admitted complaining of pain in the left hip. Physical examination showed limitation of motion on the affected side. Roentgen examination showed a bone button lying in a rarefied area in the superior marginal lip of both acetabula. We believed the condition to be either aberrant centers of ossification, as described by Pirie and others, or osteochondritis dissecans. The patient was seen a year later, complaining of pain in both hips. This was increased by abduction and outward rotation. Roentgen examination showed a partial fusion of the bone particles noted on former examination. The roentgen examination, showing bilateral separate bone fragments in hollowed out areas, which, combined with the bilateral hip pain increasing with abduction, makes us suspect that this abnormality may not be due to an aberrant epiphyseal development, but is possibly due to osteochondritis dissecans.

Cases 11 and 12.—A white girl, thirteen years old, was admitted December 6, 1928, complaining of weakness and pain in the dorsal spine. Physical examination was negative. The lateral film of the spine showed small bone fragments at the anterior superior and inferior margins of the bodies of all of the dorsal vertebrae, with small hollowed out notches in the bodies corresponding to them. These were thought to be most probably aberrant centers of ossification. The roentgen findings, which would be typical of osteochondritis dissecans if seen elsewhere in the body, together with the dorsal spine pain, has made us wonder whether these were centers of ossification, or an unusual osteochondritis dissecans. We have seen this appearance only once before, and that was in a boy fifteen years old whose spine was examined radiologically to rule out bone injury after an automobile accident. His spine presented identically the appearance of the girl previously described.

Poirier has given us an excellent description of the centers of ossification in the vertebra, and describes three centers for the body. These are, first, a central mass, which may be divided, and then later centers for both the superior and inferior surfaces. These latter centers, however, occupy a rather central position from before, backwards. The small bone buttons seen in our two cases could not correspond with these centers, as they are on the most anterior margins of the bodies. Kohler, however, reports similar findings.

A careful study of the pathologic reports on our operative cases, together with the gross appearance at operation and a consistent lack of bacterial growth from tissues from the lesions, strongly inclines us to accept Axhausen's classification. Axhausen feels that osteochondritis dissecans, together with Legge-Perthes' osteochondritis deformans, Kienbock's malacia of the carpal semilunar, and Kohler's disease of the tarsal scaphoid and head of the metatarsal, all belong under the general heading of "aseptic epiphyseal necrosis." The histologic appearance is the same in all. There is a consistent lack of bacterial growth in all, and all occur in about the same age period. These common factors are certainly highly suggestive of a common etiology. It is our hope that in the near future we may reproduce some of these lesions in experimental animals.

The writers believe that osteochondritis dissecans is more frequent than is usually believed, and that more careful study, with this condition in mind, will reveal many more cases than are diagnosed at present.

The writers wish to thank Dr. A. F. Voshell,

of the Orthopedic Department, for his kindly assistance and helpful comments, which greatly aided in the preparation of this subject matter.

SUMMARY

A study of the literature has indicated a decidedly lower incidence of osteochondritis dissecans than has occurred in our experience. This discrepancy is thought to be due chiefly to an unfamiliarity with the condition. The literature, pathology, and theories of etiology have been reviewed briefly. Nine fairly typical cases have been reported in this communication, two being bilateral, and others occurring in atypical locations. Three questionable cases are reported in an attempt to evoke helpful discussion. Finally, it is believed that osteochondritis dissecans is but one phase of the deforming non-infectious diseases of adolescence of which Legge-Perthes' disease, Kohler's disease, and sliding epiphysis are examples.

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DISCUSSION (*Abstract*)

Dr. Alfred L. Gray, Richmond, Va.—I should like to call your attention first of all to the terminology here. Bear in mind that the term osteochondritis dissecans comes from *dis* and *secare*, which means to cut off, and desiccans from *desiccare*, which means to dry up. Many people fail to recognize the difference.

It is a very remarkable thing how medical, surgical, roentgenological and pathological cases run in groups. Years ago I found a case of hernia of the diaphragm. At that time I found only two cases reported. That year I had four cases. Dr. Archer has succeeded in gathering a most unusual collection of cases of this condition in a short time. It certainly is not a rare condition, and we shall see it oftener if we bear it in mind.

Dr. Archer has reviewed the literature very completely to date, and I am sure that he, as well as I, agree with Dr. Axhausen in his theory of the etiology; that is, it is a traumatic disturbance of the arterial supply to a localized area, perhaps also assisted by infection. I am convinced that not only the conditions that he has mentioned, Kienbock's malacia and Kohler's disease, but one or two others, including osteochondritis of the dorsal spine, belong in the same category.

I mention also Kummell's disease, a traumatic destruction of the body of the vertebra, perhaps due to interference with the circulation; Perthe's disease, Schlatter's disease, Preiser's disease of the carpal scaphoid, and perhaps several others, which are named for the men who have written most about them.

There is one phase of this question, which, after Dr. Archer had written his paper, I asked him to leave for me. That is the medico-legal aspect.

I had a case sometime ago of a man who was instituting suit for damages against the Railway & Power Company, and amongst the various examinations, I discovered a lesion almost identical with one of Dr. Archer's in one hip. Suppose this patient had had an injury, and we made an examination only of that bone, the patient complaining of pain in the hip. We would almost inevitably state that that patient had fractured the rim of the acetabulum, and would testify to that effect unless we examined the other side and found the condition over there. These conditions are traumatic, perhaps, but not the result of the trauma that is under consideration at the time.

I am quite sure I have diagnosed compression fractures in young men, following a more or less definite injury, as in a football game, that were not compression fractures, but manifestations of osteochondritis of the vertebrae.

Dr. Dean Lewis, Baltimore, Md.—I am glad that Dr. Gray emphasized that this lesion is osteochondritis dissecans and not desiccans. This is a dissecting, not a drying osteochondritis.

Dr. Richardson, of Los Angeles, some years ago showed me several cases of this lesion. The number of cases that he had made me believe that it is more common than we think. It is most common, as Dr. Archer has said, in the knee, but occurs in the elbow and carpometacarpal joint of the thumb. It probably is the cause of some of the symptoms associated with the so-called internal derangements of the knee joint. We should make it a habit to view carefully the internal condyle near the intercondyloid notch in peculiar lesions of the knee joint. We may find a piece of the articular cartilage lying in a depression which resembles a casket. Such cartilages have not been as yet cast off to form the joint mouse, or floating cartilage.

Dandy has recently observed three interesting cases of cartilage in the spinal canal, which apparently had been separated from the intervertebral discs. A history of trauma in these cases may be elicited. These cartilages may cause paralysis.

Osteochondritis dissecans is a lesion which, I believe, is frequently overlooked.

Dr. E. H. Teeter, Jacksonville, Fla.—I should like to ask the essayist if he has seen cases of this type in the articulation of the maxilla?

Dr. Archer (closing).—I brought up the two cases of the spine, of the anterior superior and inferior margins of bodies of the dorsal vertebrae, because it is

not clear in my own mind whether those cases are dissecans. I have just wondered if the writers heretofore have been entirely correct in interpreting these little bone margins as aberrant centers of ossification instead of osteochondritis dissecans. I do not know whether any operative or post mortem work has been done on such cases.

I have never seen a case in the maxilla, and in my search of the literature I did not run across a reference to that condition. I do not see why it could not occur there as well as in any other portion of the bones covered with cartilage.

ACUTE PLEURAL EMPYEMA: A REVIEW*

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HISTORICAL

The earliest known reference to empyema is that of Hippocrates (460-370 B.C.), though it is probable that incision of *empyema necessitatis* was practised by the Chinese and Egyptians.

Hippocrates taught that light infections of empyema recover without any surgical intervention. In the more severe cases a thoracotomy should be done by knife or cautery or a rib trephined and the pus evacuated on the fifteenth day, not before. After the pus was gradually evacuated, a mixture of warm wine and oil was injected. He gives a good prognosis when the pus is uniformly white, free from mucus, and not foul smelling, and a fatal result is prophesied when the pus is greenish or dark colored, contains mucus, and particularly when it is foamy.

Euryphon of Knidus, a contemporary of Hippocrates, treated empyema with the cautery, and tells of lives saved by this method.

Aristotle (384-322 B.C.), who taught anatomy by dissection, writes of the treatment of empyema.

Pliny (23-79 A.D.), whose "Natural History" includes much medicine, tells of a soldier with empyema. He was despaired of by his physician and sought relief by death in battle. He received a wound in the thorax, the pus escaped, and he recovered.

Celsus, at the beginning of the Christian era, wrote as follows: "Fistulae of the chest are very difficult of treatment, so that sometimes physician, sometimes patient, giving up hope, leaves the case to nature herself."

Aretaeus, the Cappadocian, in the Second Century, gives a classic account of pleurisy with empyema.

Galen (131-201 A.D.), the greatest Greek physician after Hippocrates, and founder of experimental physiology, began practice in Rome in 164 A.D. He emphasized the importance of providing for the escape of pus in the thorax. He removed portions of carious ribs, injected warm wine into the cavity and urged the patient to cough while leaning toward the affected side. He also used honey and water in empyema cavities.

Paul of Acgina (625-690), the last of the Greek eclectics, and author of an Epitome of medicine in seven books, was a good surgeon, but stopped short of opening the chest for empyema.

Avicenna (980-1037), Albucassis (1013-1106), and Avenzoar (1094-1162), approved of paracentesis and the cautery in empyema treatment.

Guy de Chauliac (1300-1368) was the most eminent surgical authority of the Fourteenth Century, and physician to the popes at Avignon. He defines empyema as follows: "Empyma, or empyema, in Greek signifies a collection of pus in whatsoever part of the body it may be, but more properly used meaning a collection of suppurative matter in the testes, thorax or abdomen. In a more restricted sense it means pus in the thorax: this is the most proper and common significance. Following it one calls those who have pus in the thorax 'empyes' or 'empyiques' in Greek, and suppurants or purulents in Latin."

Lanfranchi (?-1315), of Milan, was a cauterist and as such was averse to the knife. However, he did not hesitate to operate for empyema and wounds of the intestines.

Andreas Vesalius (1514-64), professor of anatomy at Padua, performed a successful operation for empyema upon Don Carlos, of Aragon, son of Philip II, of Spain. He reports two other successful cases and insists that an incision should always be made for empyema, and that no one should die of the disease.

Empyema was also treated by Fabricius, of Acquapendente (1537-1619), who succeeded Fallopius as professor of anatomy and surgery at Padua. Another eminent advocate was Ambroise Pare (1510-1590), a lowly dresser at Hotel Dieu, before gaining fame as an army surgeon.

The writings of Scultetus (Johann Schultes 1595-1645) are the first reference in the French language to empyema. He discusses indications, time, site, technic and instruments to be used in paracentesis. Fienus is the earliest known writer in German. He discusses the problems involved in pneumothorax after operation. Ingram is the author of the earliest available treatise in English.

Kisnerus wrote that venesection, generally a most excellent remedy, is almost supreme in cases of empyema.

The greatest impetus to surgery of the chest was the description of percussion by Auenbrugger, of Vienna, in 1761. Stoll, Corvisart and Laennec were the first to emphasize its value in the diagnosis of empyema.

As late as 1825 Dumont advises mercury, bleeding, purgatives, and a rigid diet, adding that the most common termination is death. Laennec is reported as saying that the operation for empyema is rarely successful.

Dupuytren (1777-1835) reported 50 cases with only four cures. He was an ardent advocate of thoracotomy, but when he, himself, developed a fatal empyema he refused operation, saying that he preferred to die by the hand of God than by the hand of the surgeon.

In 1881 Homen published a series of 91 cases treated by seven surgeons between 1868-1876. Of these, 47 per cent were cured, 23 per cent had fistula, and 29 per cent died. In another series of 141 cases there was a 33 per cent mortality.

The first rib resection for drainage was done by Walter in 1860. In 1876 Bulau, in Germany, introduced his method of intercostal drainage. In the same year a similar method was described by Hewett in England.

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†Dr. Lyle B. West was killed in an airplane accident on February 24, 1930, while enroute to a patient seriously injured in an automobile accident in Florida.

Estlander, in 1879, reported six cases of obliteration of chronic cavities. In 1890 Schede described the mutilating procedure which bears his name. Delorme, in 1892, suggested his decortication method which was first done by Fowler in 1893.

Perthes, in 1898, recommended suction by the use of Bunsen's air-pump attached to a hydrant, but this was found to use too much water.

MORTALITY AND COMPLICATIONS

Modern methods in the treatment of empyema may be said to begin with the hitherto unprecedented epidemic of 1917-1918.

Wilensky reports a mortality of 28 per cent in 299 cases at the Mt. Sinai Hospital (N. Y.), during the years of 1904-1914. Lilienthal and Ware in 1916 reported a mortality of 21 per cent in 38 cases among children under twelve, and a mortality of 20 per cent in 25 cases in adults. Other pre-war statistics vary from 18-55 per cent.

It has been noted by some that prior to the 1917-1918 epidemic, empyema was not a common complication of pneumonia. In adults, lung abscess was a rather more frequent sequella.

At the time of this epidemic the generally accepted treatment was early open drainage. Such treatment resulted in the following mortality: Camp Funston 84 per cent, Camp Wheeler 65 per cent, Fort Riley 63.1 per cent, and Camp Lee 48 per cent. At Camp Dodge 45 per cent of those with pneumonia had empyema as a complication, and of these there was a mortality of 64.8 per cent. At Fort Riley 21.2 per cent of the lobar pneumonias were followed by empyema, and early open operations in these gave a mortality of 63.1 per cent. With aspirations and later operation, the mortality was reduced to 22.3 per cent. At Camp Pike, from September, 1917, to September, 1918, there were 1,285 cases of pneumonia and 9 per cent of these developed empyema. Sauerbruch reports a 90 per cent mortality in German troops treated by rib resection in 1918-19.

The average mortality as determined by the Surgeon-General's questionnaire of Feb. 21, 1918, was 30.2 per cent.

The work of Graham and Bell, of the Empyema Commission, proved that the large majority of the deaths during the epidemic were due to the establishment of open pneumothorax in the absence of adhesions and at a time when the vital capacity was greatly reduced by the pulmonary disease. The load on the respiratory and cardiac systems was too great for compensation.

When the treatment was changed, according to these findings, the mortality was greatly reduced. At the hands of the Empyema Commission, for example, it reached the very low figure of 4.3 per cent. It was the opinion of this commission that practically no cases of non-tuberculous empyema, uncomplicated by lung abscess, suppurative pericarditis, so forth, would die if treated in accordance with these principles.

Statistics since the great epidemic show only slight improvement. Demel, of von Eiselsberg's clinic, reports 141 cases in ten years with a mortality of 30 per cent. Downes reports 446 cases in nine and one-half years with a mortality of 29 per cent, an operative mortality of 17 per cent, and an infancy mortality of 58 per cent. Graham shows a mortality of 11 per cent in 34 cases, Todd and Cutler 12 per cent in 154 cases, Farr and Levine 19 per cent in 371 cases, Scott 40 per cent in 26 cases, Wilensky 28 per cent in 200 cases, and Bohrer 16.8 per cent in 154 cases. He has a mortality of 10.9 per cent in children from two to six years of age and in those from six to twelve years 3.3 per cent. In this age there has been repeatedly noted the smallest mortality.

The mortality of childhood empyema is about four times greater in the first than in the fourth year, and it is almost always a complication of pneumonia. Death of children is usually due to a general condition of debility, brought on by the previous illness, or due to existing complications and not directly to the empyema itself.

Sauermondt, of Leyden, reports 185 cases of empyema treated by open methods with a mortality of 11.3 per cent. Pogorschelsky, of Dortmund, reports a mortality of 60 per cent in infants and 26 per cent in young children. The mortality of Johns Hopkins, 1915-28, in children under two years of age, was 29 per cent with rib resection and 50 per cent with closed drainage. McEnery and Brennemann report an operative mortality of 25 per cent and a mortality, using aspiration only, of 9 per cent. Cameron, of Guy's Hospital, reports a 70 per cent mortality in infants less than one year of age and 50 per cent in those from one to two years. Spence has a mortality of 64 per cent in infants under one year and 45 per cent in those under three years of age.

Hart reports a 10 per cent mortality in 50 cases treated by his tidal irrigation method. The 1928 mortality of the St. Louis Children's Hospital was 2.7 per cent in 36 cases treated by aspiration, with rib resection only when necessary. Operative mortality is very great in in-

fancy, as witness the following: Ziebell 75.6 per cent, Finkelstein 72.5 per cent, Hodge 74 per cent; with punctures alone, Zeibell 52.6 per cent, Finkelstein 32 per cent, and Holt 26.6 per cent.

Empyema is a more frequent complication of pneumonia of children than of adults, and at least 75 per cent of childhood empyemas are pneumococcic. Bisdom finds that 4-5 per cent of the pneumonias of children have empyema as a complication, while Fraser, from statistics of the Edinburgh Children's Hospital, gives an incidence of 21-24 per cent.

Empyema occurs most frequently in infancy and its incidence gradually decreases with age. Complications also occur more frequently in children.

Hart found that 75 per cent of his cases had complications as follows: pneumonia, osteomyelitis of ribs, bilateral empyema, lung abscess, bronchial fistula, pericarditis, septicemia, peritonitis, meningitis, otitis media, mastoiditis and pyelitis.

Bronchial fistulae are most commonly found as a complication of this disease, but the majority close spontaneously. Hart had seven in 50 cases, but all closed with his tidal suction method.

A parapneumonic empyema is more serious than a metapneumonic one. The former is particularly serious if it develops at the peak of the pneumonia from a ruptured cortical focus. The more rapidly the exudate increases, the more severe are the manifestations. The parapneumonic type is characterized by an early massive sero-fibrinous fluid and is usually due to the streptococcus. The pneumococcus infection causes a much slower fluid formation.

Empyema in the upper portion of the interlobar fissure is technically the most difficult, but fortunately is rare.

Mesial empyema is one which is bordered internally by the mediastinal pleura, anteriorly by the lung hilus and externally by the visceral pleura. A supraphrenic one is more apt to progress toward the median side than toward the lateral chest wall. The mesial variety is seen infrequently, and usually as a secondary condition. Persistence of fever after adequate drainage suggests undrained foci.

The most favorable prognosis is given by a pneumococcus infection in pure culture. Infections of streptococcus, staphylococcus, and influenza bacillus give a more serious prognosis.

Recent bacteriologic work on the pneumococcus is interesting. Cooper, Edwards and Rosenstein have separated ten distinct groups within type IV pneumococcus. A monovalent serum can be prepared by each of the ten groups and there is very little cross protective power against the other groups.

Griffith has recognized two variants of pneumococci. The S (smooth) forms are virulent, they produce the specific soluble substance upon which type specificity depends and they form colonies which have a smooth surface by reflected light. The R (rough) forms are avirulent, they do not produce the specific soluble substance and they form colonies which have a rough surface. The two forms are found in types I, II and III, and in types II and III they can be converted *in vitro* and *in vivo*.

These findings may account for the great variance in virulence at different seasons and localities and may offer a more specific treatment for the future.

METHOD

The history of empyema shows that remarkably little progress in treatment has been made in the twenty-two centuries of which we have record.

The literature on the subject is voluminous and many opinions and methods of treatment are given.

Many mechanical devices have been advocated and since the advent of antisepsis many chemotherapeutic agents have been used.

Morgenroth first showed the specific action of optochin ("best cinchonin," ethyl hydrocuprein hydrochloride) on the pneumococcus. It is used in pneumococcus empyema by many with good results (Gralka, Worringer, Kolmer and Sands, Bussell and Hirszfeld, Lowenburg, Grosser, and Leitner).

The usual technic is irrigation with a 0.5 per cent solution of optochin, leaving a 5 per cent solution in the cavity (0.025 gm. of optochin per kg. body weight). Failures with this method are accounted for by the presence of the malignant smooth form of pneumococcus, which is resistant to optochin. A case is reported of the reversion, by the use of optochin, of the resistant smooth form to the non-resistant rough form.

Morgenroth has also shown that "vucin" (isocytyhydocuprein) is actively bactericidal for staphylococcus, streptococcus and gas bacillus infections, but as yet its use is in the experimental stage.

In European clinics Pregl's (hydriotic acid, iodine and iodic acid) and Kermannsdorfers' solution (pepsin 20, phenol 2, hydrochloric acid 2, water 400) are used in the pleura.

Churchman has shown that gentian violet solution has a bacteriostatic effect on the Gram-positive organisms and this has been used with success on pneumococcus and streptococcus cases.

Major reports a series of 57 cases treated by aspirations and gentian violet. There were 45 recoveries by this method alone. From four to fourteen aspirations were necessary in each case.

The use of Beck's paste is advocated. Beck reports that 80 per cent of his suppurations healed with this agent. Unger fills the cavity with paraffin with good results except for a sense of heaviness.

Rib trephining has been given up, but few advocates remain. Autovaccinotherapy supplementing drainage has its advocates.

J. B. Murphy used 2 per cent formalin in glycerine in cavities with good results, though painful. A weak solution of tincture of iodine in warm water is used by some.

Repeated aspiration is used by many with good results, particularly in infancy. Many empyemas have been cured by a single puncture, though the majority require repeated aspirations. Of the hemolytic streptococcus cases seen by the Empyema Commission, 13 per cent recovered by aspirations alone and without operation. Harloe reports a 7.5 per cent mortality in nursing children with puncture alone. McEnery and Brenneman report a 9 per cent mortality in 33 cases in children with aspirations alone, averaging four aspirations per case.

Passeron advises evacuation of the pus through a thoracotomy under water with the patient in a bath tub.

The suction apparatus of Potain and Dieulafoy have been used in the closed methods. McKesson has recently described an adjustable electric suction pump for postoperative drainage.

Dakin's solution, brought out in 1918, is very popular, but many large clinics now use simple saline irrigations with equal success in the acute forms. Stevens has shown that Dakin's solution should have a free chlorine content of 0.4-0.5 per cent. If it is too alkaline the exudate and scars are absorbed too quickly, thereby increasing the likelihood of hemorrhage and bronchial fistula. If the chlorine content is below 0.4 per cent the sterilization is faulty.

Sauerbruch and Hart report the successful treatment of tuberculous empyema with the closed siphon method.

The pneumatic chambers of Sauerbruch and Willy Meyer and the increased intratracheal pressure methods were useful in early open operations.

A silver cannula was early advised. Demel uses a tubular instrument shaped like an ice tong, which is put into the pleura through different intercostal spaces. When closed it cannot slip out. A simple tracheotomy tube has been used. Bouvier inflates a rubber balloon in the thoracotomy opening around the drainage tube. A valve allowing pus to exude, but no air to enter was an improvement used by Mikulicz and Thiersch. A valvular thoracotomy as described by Aupepin (1810), having the skin and pleural openings at different levels, is in use.

Mozingo, of the Walter Reed Hospital, repopularized in this country the closed method of irrigation during the influenza epidemic. He reports 114 cases with three deaths.

Hart has developed a theorectically ideal system of closed tidal irrigation, in which the irrigating solution (saline) ebbs and flows through the cavity to a lower receptacle.

There are two factors of primary importance. The pus demands free drainage, and the lung abhors collapse, due to open drainage. It has been shown that pleural resistance is lowered by atmospheric pressure.

Conceptions have changed from pure anatomical considerations of the disease to those of function caused by open methods. The suppurative pleurisy is not the ordinary abscess which calls for rapid drainage.

Hedblom says:

> "The cardinal principle is adequate drainage by any method, but infancy, old age, acute streptococcus empyema, or a critically ill patient of any age requires the closed method of drainage in the early stages."

Empyemas of childhood are more serious because of the greater flexibility of the mediastinum and here conservative treatment is especially indicated.

The disastrous results of early operation in influenzal empyema is the most important observation on the subject and closed drainage the best therapeutic lesson learned. If the vital capacity is so low as to approximate the tidal air, even a small opening may be fatal.

The methods in use may be listed as follows:

(1) Intermittent aspiration.

(2) Small intercostal thoracotomy with closed drainage.

(3) Intercostal thoracotomy with open drainage.

(4) Large intercostal thoracotomy for exploration, later sutured for closed or open drainage.

(5) Thoracotomy with resection of one or more ribs.

(6) The physiologic operations of Willy Meyer, Sauerbruch and Pickhardt.

(7) Filling the cavity with Beck's paste or paraffine.

METHODS PREFERRED

With equally good results, the simplest method, that with least shock and deformity, must appeal to all.

In the writer's opinion, simple repeated aspirations should be given a fair trial in all patients up to three years of age. The aspirations are done every three days, or oftener if indicated. The needle before removal should be washed with an antiseptic solution. And while being withdrawn this solution should be deposited along the needle tract.

In those cases which do not yield to aspiration and in all older than three years the closed siphonage drainage is preferred.

The early establishment of continuous drainage with negative pressure has the advantages of keeping the pleural cavity relatively free from liquid exudate and organisms, of aiding the expansion of the lung as well as of avoiding an open pneumothorax with its attendant dangers.

The thoracotomy, when possible, should be done along the diagnostic aspirating needle left *in situ* in the eighth or ninth interspace in the posterior axillary line. This can be done in the patient's bed without pain by local anesthesia. Subsequent pain will be diminished if novocaine followed by alcohol is injected along the intercostal nerves posterior to the thoracotomy wound.

A bistoury is pushed in beside the needle, then a trocar and cannula are pushed along this wound. An 18-22 F. soft rubber catheter, lubricated with K-Y ointment, is then put through the cannula immediately on removal of the trocar. The catheter should fit the cannula as snugly as possible and requires a lubricant to insure its being tight in the thoracotomy wound when the cannula is withdrawn. A three inch square of heavy rubber tissue with a hole in its center is slipped over the catheter to the skin surface and held fast with vertical and horizontal adhesive strips. No pin is used in the catheter, for this gives a possible entrance of air. By keeping the catheter clamped there is very little loss of fluid.

The skin should not fit tightly around the catheter, but should allow for the escape of infected fluid should this occur.

The catheter is now connected with a Y tube, having one arm connected to a suspended irrigator, and the other connected to empty under the surface of a bottle of sterile boric solution on the floor. The irrigating solution (saline) can be let into the closed cavity at intervals by releasing cutoffs.

If the connecting tubes from the irrigator to the pus receptacle are made to describe a small curve, the former below and the latter just above the thoracotomy opening, a fluid trap is formed. Pus will overflow with a deep inspiration and the resulting negative pressure will draw clean irrigating fluid into the cavity. This has been used by Buchner, of the Children's Hospital, with success. It is simple to set up and simple to care for on the wards. The patient is allowed some freedom of motion.

This method will not effect a complete cure in every case, but in that small percentage of cases, the persisting cavity will be so small that a collapsing operation will not be a deformity.

Open operation with foul smelling pus saturating many dressings daily is insanitary, appetite destroying, and expensive.

The diet should be liberal. Bell has shown that unless attention is paid to the diet, patients will have a nitrogen waste of as much as 21 gm. a day, 15 gm. being the healthy normal. Such protein destruction can be saved by increasing the carbohydrate intake. Lactose, given in fruit juices, is well taken, as it does not have the sickening sweet taste of cane sugar. The diet should be of higher caloric value than in health, up to 4,000 calories.

In conclusion, the writer does not insist on the above method to the exclusion of all others. In our hands it has been most satisfactory, and it has given lowered mortality figures in the hands of others. However, in all fairness, one cannot choose an ideal treatment from mortality statistics because conditions vary; virulence, season, the age of the patient, and the various infecting organisms must be considered, in addition to the mode of treatment.

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DIMINISHING MORTALITY AND MORBIDITY*

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"The novelty of today somehow becomes the commonplace of tomorrow" (Flexner).

"The standard bearers of medicine at all times overcame obstacles with courage, persistence, keen observation, sincerity of purpose and humility" (A Psalm of Life).

These characteristics have contributed largely in the effort of pioneers in medicine to reduce mortality and morbidity.

America has contributed its quota of men who during the Nineteenth Century helped to diminish mortality and morbidity. It is true that France produced Pasteur; England, Lister; but America produced Long, Morton, McDowell and Sims, and at a later period Crile and Cannon added to our means of treating that dreaded and almost certainly fatal condition known as shock.

Matas, Ochsner, Murphy, the Mayos and Crile have each contributed to the present development of surgery.

The Twentieth Century has not been laggard in its appreciation of its obligation.

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There are three great factors to be considered in any effort to reduce surgical mortality: (1) the surgeon; (2) the procedure; (3) the patient.

When we consider the first of these factors at times we are inclined to think that an appreciation of the difference between a surgeon and an operator is a modern conception. This conclusion is not justifiable when one looks back over the long list of great teachers in the history of the development of surgery.

The moral principles of such great leaders as Hippocrates, Guy de Chauliac, Namonides have come down to us through the ages.

A little more than one hundred years ago, John D. Godman of Philadelphia, in an editorial note in a manual on surgical operations of Lisfranc, compared the surgeon and the operator as follows:

"The surgeon reluctantly decides on the use of instruments, the operator delays no longer than to give his knife a keen edge. The surgeon is governed by the principles of medicine, the operator most generally by the principles of interest. The surgeon is an honor to his profession and a benefactor to his fellow creatures. The mere operator renders the profession odious, and is one of the greatest curses to which mankind, among their manifold miseries, are exposed."

We see from the above that the ethics which are the guide of the American College of Surgeons in its efforts to have surgery used only when necessary, and then by the most approved methods, is not a new service, but is simply attempting to carry out those noble principles which have come down to us as our natural heritage from the great leaders and teachers in medicine.

Let us postulate then that early diagnosis through careful history taking and examination, followed by a proper interpretation of signs and symptoms, will lead to a lessened morbidity and mortality when such procedures as our present knowledge of pathology indicates, are adopted. As a corollary it should be added that the difference between surgeons lies not altogether in the amount of information possessed, but in the relative care each exercises in arriving at a diagnosis before surgery is undertaken. With this preamble let us approach the subject.

In a paper of this length one can only approach some of the many aspects of this problem, and touch only lightly on each. We shall have to content ourselves with a bird's-eye view of the development in surgery during recent years.

In order to do this briefly, I shall have to discuss the subject under the following headings:

(1) Traumatic cranial surgery.
(2) Thoracic surgery.
(3) Abdominal surgery.
(4) Surgery of the extremities.

HEAD INJURIES

Cranial surgery based upon scientific investigation and knowledge of the third circulation is largely a development of recent years and may be associated with the names of Victor Horsley, William Mac Ewen and Harvey Cushing of Boston. To the last named, an American surgeon, may be given the credit for having done more to develop the art and science of cranial surgery than any one else. The great number of men whom his life and work has stimulated makes America today probably the leading country in cranial surgery.

Head injuries must always be considered as serious until proven to be otherwise. Repeated blood pressure readings should be made and correlated with the pulse. Steadily diminishing pulse rate means increasing intracranial pressure. Spinal manometric readings are of the utmost value in determining the existence of increased intracranial tension. The finding of bloody spinal fluid after an accident is indicative of subdural bleeding. Where the classical syndrome of middle meningeal hemorrhage does not exist one may with propriety make use of repeated spinal tappings, magnesium sulphate, by mouth or by rectum. If there is pure blood in the spinal taps, it may be advisable to discontinue tapping and operate, if there is localization. This, however, is best done after clot formation.

The dilated pupil is of the greatest value in these cases, as it is found most frequently on the side of the subdural hemorrhage. Carter states:

"When a pupil is large on admission and comes down to normal the prognosis is apt to be favorable, but when a pupil which was equal to its mate on admission later becomes dilated, we feel that intracranial pressure is progressing and become suspicious of an extra or intradural hemorrhage."

These cases cannot be catalogued absolutely because in many of them more than one type of injury is present. Indiscriminate operating should be avoided and most cases should be allowed to react from the initial shock before operating. The majority of head injuries except depressed fractures, middle meningeal hemorrhage and subdural clots, should be treated by conservative means.

THORACIC SURGERY

Progress has been made during recent years in the surgical management of thoracic conditions. Prior to the great influenza epidemic in 1918 large pleural accumulations were treated by thoracotomy with a resultant high mortality. This was due in part to collapse of the lung on the opposite side. The encroachment on the opposite lung field was shown by Evarts Graham and others to be due to the absence of adhesions. Their work has resulted in the use of conservative management of these cases. Where there is an accumulation of nonpurulent fluid in the pleura, repeated aspiration should be done for the purpose of relieving dyspnea. When frank pus is found thoracotomy may be done with safety. Adhesions which are present prevent displacement of the opposite lung.

Lung Abscess.—Lung abscess about which little was generally known until recent years is now not uncommonly seen to respond in a spectacular manner to surgery. Two of the etiologic factors, in particular, should be mentioned: aspiration and blood stream infection. Aspiration is of importance as an etiologic factor because of the frequency of tonsillectomies and other operations on the throat. Where proper attention is paid to suction during these operations the number of post-tonsillectomy pulmonary abscesses is exceedingly small.

Abscess of the lung due to aspiration of a foreign body is best treated by bronchoscopic operation and drainage. Peripheral abscess cannot be so treated. Surgery should be resorted to after proper medical regime (postural drainage) has been tried out and satisfactory improvement has not resulted.

Accurate localization by stereoscopic x-ray and lateral projection (Le Wald) should enable the surgeon to make a more direct approach.

Another valuable point obtained by the use of the lateral projection is a fluid level which is at times shown and this is altered by the patient's change of position.

After the decision has been made to operate, the following, I believe, are important considerations:

(1) Approach the abscess by the most direct route.

(2) Use local anesthesia.

(3) Avoid aspiration through the pleura. (This procedure may infect the pleura which would otherwise not become involved and then an empyema will be superimposed upon the lung abscess.)

(4) The abscess cavity should not be opened until one has satisfied himself that there are adhesions between the pleura and lung. (When a rib has been resected the absence of adhesions can usually be determined by inspection. If the lung movement can be seen through the pleura, packing may be resorted to and forty-eight hours later the cavity incised either with the cautery or knife.)

(5) Suction should be avoided, as this may cause bleeding. Gauze packs should be resorted to only if there is hemorrhage.

Surgical treatment of tuberculosis, including phrenectomy and other measures intended to immobilize the affected lung, need only be mentioned here to indicate surgical progress and invasion of fields formerly sacred to the internist.

ABDOMINAL SURGERY

In this field I should like to direct attention to the great progress which has been made in diagnosis and management of diseases of the spleen and some of the great crises which occur within the abdomen. A better understanding of the reticulo-endothelial system has led to spectacular results from splenectomy in splenic anemia, hemolytic jaundice and purpura hemorrhagica. It is true that indiscriminate operations, in cases in which accurate differential diagnosis has not proven the indication, may bring grief to the surgeon and to the patient. In diseases in which the spleen may be considered as taking a sufficiently active part to warrant a splenectomy there are certain essential phenomena which are of importance in differential diagnosis; blood platelet counting, red cell resistance, retraction of the clot and the differential cell count. It would be out of place at this time to go into details with reference to these particular tests.

When considering the great abdominal crises which the surgeon faces at times, perforation of the stomach and duodenum should occupy a front rank. By proper utilization of the x-ray for diagnostic information in these particular crises a distinct advance has been made.

The evidence of spontaneous pneumoperitoneum is diagnostic. No delay is entailed in order to obtain this information. The patient should be taken to the x-ray department while preparations are being made in the operating room. Immediate operation with closure of the perforation

is all that is necessary in most cases. This is certainly true if the closure does not interfere with the lumen of the pylorus.

Recovery in these cases is largely dependent on the time element. The earlier the patient is operated upon following the perforation, the less will be the mortality and morbidity. The factors of safety are increased if the surgeon avoids procedures which may be calculated to prolong the operation.

SURGERY OF THE EXTREMITIES

Progress in the management of diseases involving the extremities has kept pace with that noted in other parts of the body.

I have elected to mention only three conditions for your attention.

(1) Erysipelas.

(2) Diabetic gangrene.

(3) Gas gangrene.

Erysipelas—The *Streptococcus hemolyticus* was first identified in 1883 by Fehleisen. From that time until 1925 almost every conservative form of treatment was used. Douglas Symmers and Kenneth Lewis, in a report based on a study of 15,277 cases treated at Bellevue Hospital, stated that "none of the methods seemed to shorten the course of the disease."

In 1925, Birkaugh stated that he found that 91.2 per cent of all strains of erysipelas streptoccocci fell into one group antigenically. He then proceeded to immunize a donkey after the method of Dochez. Within the first year Birkaugh treated 60 cases with antitoxin treatment with astonishing results.

In 1927, Symmers and Lewis stated: "In Bellevue we are now using the antitoxin treatment to the exclusion of other methods." In 131 cases treated, they reported, "results that have been remarkable."

It is advised by those authors that the intramuscular route be used exclusively. Intravenous use of the Birkaugh serum has resulted in death in at least one case. Symmers and Lewis advocate a dose of 25 c. c. of unrefined antitoxin on admission and this is repeated at twenty-four hours intervals "until the erysipelas blush disappears, the edema is dissipated and the temperature normal."

The mortality in their cases has been reduced from 40 to 10 per cent in body erysipelas, and in facial erysipelas the mortality has been reduced from 6.5 to 4.5 per cent. Eighty-five per cent of the cases have been cured in from three to seven days.

DIABETES

Diabetes has become increasingly a surgical problem since the great physiologic discovery of Banting and his associates.

The enthusiasm of such men as Joslin and Lemann have so far perfected the management of the diabetic that today we are told that insulin has placed a new responsibility upon the surgeon.

We all realize that the diabetic was formerly looked upon with fear and trembling when surgical problems, such as gangrene, presented themselves.

Gangrene when associated with diabetes should not be considered as a separate entity; arteriosclerosis is the background. Diabetes merely causes gangrene to arise at an earlier period than in the non-diabetic.

Dan Jones says that at least 25 per cent of all diabetics become surgical problems at some time.

In order properly to manage these patients special attention and close cooperation between the internist and surgeon is necessary. The internist should direct all treatment except surgical technic.

Better results will be obtained in the management of disturbance of the extremities in the diabetic if prophylactic care of the circulation is provided. This includes protection against undue exposure to sudden changes of temperature, vascular gymnastics and early careful attention to the slightest wounds to avoid infection.

In 1853, Maisonneuve described a case of gas gangrene. Salleron, in 1854, reported 65 cases which he had observed during the Crimean War. During the period between 1871 and 1880, Bottini, Pasteur, Koch and Gaffky wrote extensively on the subject. It was not until 1891 that Welsh isolated a pathogenic organism from human blood.

Little attention was paid by the general profession to this condition until the War, and even after the War the condition was considered one to be expected largely in war surgery. With increasing experience, it was recognized that the disease is not uncommon in civil life. An appreciation of its importance has resulted in preparation of gas gangrene serum which when given early and in association with proper surgery has given spectacular results. The prophylactic use of this serum is bound to diminish the frequency

of the disease as antitetanic serum has diminished the incidence of tetanus.

We should be stimulated by every new advance to utilize those methods which have been proven of value in reducing suffering.

If I have said enough to stimulate the use of any or all of the procedures mentioned for diminishing morbidity and mortality the purpose of this paper will have been accomplished.

1522 Aline Street.

DISCUSSION (*Abstract*)

Dr. J. Shelton Horsley, Richmond, Va.—Most of the progress in surgery in the last twenty years has been due not so much to the improvement in mechanical technic as to a better consideration of the biological features that are involved in every surgical operation. I do not wish to minimize the importance of a satisfactory surgical technic, but the value of the proper consideration of the biologic features, meaning the physiologic and pathologic changes, in these patients is of more importance. For illustration, we may consider the operation on the thyroid gland for hyperthyroidism. Technically the modern operation from an anatomical standpoint is far less admirable and less beautiful than the operation that was done twenty years ago when the vessels and nerves of the neck were demonstrated and an extensive dissection was made. Now, however, by preparing the patient properly, exposing as little of the tissue as possible, doing the operation gently and if necessary in stages, the mortality rate has been greatly diminished from what it was when the operation was technically more satisfactory.

These improvements have been built up by considering the biologic conditions that existed in a hyperthyroid patient and trying to combat the injurious tendencies.

Formerly we purged patients in order to make the intestines flat and easy to handle. Now we know that purging dehydrates the patient, eliminates helpful secretions, and is really harmful even though it renders the abdomen flatter.

After stomach operations it is important to give rest to the stomach. Physiologic rest in all conditions is our greatest therapeutic resource. When the gastrointestinal tract cannot be used satisfactorily for absorption of food, intravenous injections of dextrose in Ringer's solution can be kept up continuously for days.

These few instances show a modern tendency in surgery toward studying the biologic conditions of the patient and meeting them, rather than glorifying too much the mechanical technical features.

Dr. I. M. Gage, New Orleans, La.—A man who takes up surgery as a specialty should spend three years as an apprentice to a recognized surgeon, dividing his time between the experimental laboratory and the laboratory of pathology.

Willis, several years ago, showed that the mortality from appendicitis, gall-bladder disease, diseases of the stomach and disease of the thyroid glands in ten-year periods was rapidly increasing. The mortality from thyroid disease had increased 175 per cent.

Over 95, almost 100 per cent of all the wool clothing we wear harbors the gas bacillus. I have shown in our laboratory that the majority of cases of gas bacillus infection following gunshot wounds, result from contamination of the clothing.

Dr. J. L. Crook, Jackson, Tenn.—Many of you remember at the last meeting of the American College of Surgeons tremendous emphasis was placed on the subject of traumatic surgery, one entire day being given to it.

We have had imposed upon us as staff members of hospitals and as part owners of hospitals a tremendous burden due to highway casualties. I have only to mention that last year 33,000 lives were sacrificed in automobile accidents alone and there were 1,300,000 severe injuries on the highways.

We can reduce these deaths and lessen the morbidity of serious injuries by seeing to it that our hospitals are properly organized to bear this tremendous burden. Any factor in the organization of the staff to give more competent service in emergency casualties would be a great factor in reducing death and lessening morbidity. As one important factor: all modern hospitals should have a fully equipped operating room on the first floor, with immediate access from the outside world, with a constant temperature of 80°, available for twenty-four hour service. We all know, who have had practical experience, that one of the greatest helps in saving the life of a patient is the favorable environment in which the operation is performed.

The family physician largely determines the issue in acute abdominal cases. The fact that we have competent, able family physicians surrounding the modern hospitals is largely responsible for the fact that we are saving many abdominal casualties, where formerly we lost them. These physicians should place the responsibility of acute abdominal conditions upon the shoulders of the surgeons. The mortality of appendicitis is over 10 per cent. It should not be 1 per cent. Delay in bringing the patients to the surgeons is the largest factor in the mortality.

Therefore, to summarize: an efficient physician, a modern highway, a well equipped hospital and common sense and technical skill on the part of the surgeon will all be required to reduce the mortality and morbidity.

Dr. J. T. Nix, New Orleans, La.—It is most important to determine the surgical risk, whether the patient should be operated upon at all. Patients presenting surgical conditions are not always surgical risks, and they should be made good surgical risks before being operated upon, if it is possible. The work is essentially the combined one of the internist and of the surgeon.

We cannot lay too much stress on preliminary laboratory examinations. Determination of carbon dioxide reserve and other blood chemical estimations will often save us trouble after operations. If a condition of alkalosis or acidosis is present or impending, it will respond satisfactorily to its appropriate treatment.

Blood transfusion should be thought of every time that we consider a serious operation. No extreme major operation should be attempted until donors are available. The patient should be typed before operation, or blood from prospective donors should be matched. Transfusion of blood should be done during and immediately following the operation when it is indicated. Very often repeated small blood transfusions are the only means of building up a patient for an operation and making him a good surgical risk.

Diathermy should be available. There are many operative procedures in which diathermy would be distinctly beneficial, during the operation or immediately afterwards, especially long operations with much exposure, handling, and chilling of viscera. I recently observed a case where total suppression of urine of twenty-four hours' duration was relieved promptly by diathermy.

Dr. Cohn (closing).—This paper was intended to stimulate each one of us to do a little better in whatever we attempt to do.

Dr. Nix mentioned the surgical risk. I intended to cover that subject in the part of the paper in which I said, "Careful examination and careful history will lead to the utilization of those methods best calculated to benefit the patient."

THYMUS*

By DOUGLAS D. MARTIN, M.D.,
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So much controversy has arisen during recent years in the study of the thymus that it will not be my effort to establish new theories nor any one fact positively. I shall attempt to review certain well known facts (many facts about the thymus are disregarded), and, if possible, define more accurately the conclusions which are being formulated.

The thymus at birth, according to Hammar, in Osler's *"Medicine,"* weighs 12.0 grams.

From one to five years it weighs 23.0 grams.

From six to ten years it weighs 26.0 grams.

From eleven to fifteen years it weighs 37.5 grams.

From sixteen to twenty years it weighs 25.5 grams.

After twenty years there is gradual atrophy. In a review of the literature, Short, of Harvard Medical School, stated the conclusions of Mosher as follows:

(1) The thymus is not necessary to life.

(2) Thymectomy has no effect on the growth and development of the skeleton and organs.

(3) Castration delays the involution of the thymus.

(4) Thymus feeding to salamander larvae with parathyroids causes tetany.

(5) A substance contained in the thymus, when injected, causes convulsions.

(6) Thymic hyperplasia follows double suprarenalectomy.

(7) Thymic hyperplasia in thyrotoxicosis is secondary.

(8) In birds a relationship exists between the thymus and the egg producing mechanism.

(9) So-called "thymic death" is not due to tracheostenosis and probably bears no relation to the state of the thymus gland.

(10) One of the functions, if not the primary function, of the thymus, is to produce leucocytes.

(11) The thymus may also have an antitoxic function (Hammar).

The evidence, on the whole, is that the thymus is not a gland of internal secretion. That it may serve as a manufactory and a storehouse for lymphocytes, at the time of life when there is the greatest need to combat infection, seems to be a popular view.

For some years it has been the routine in certain hospitals to examine all children radiologically before submitting them to a general anesthetic. If there is found what is thought to be an enlarged thymus gland, a prophylactic roentgen treatment is given because of the rather well entrenched belief that an enlarged thymus gland is an integral factor of the syndrome of the so-called status lymphaticus, a reputed cause of otherwise unexplained death. This, I believe, is a wholly uncalled for procedure, which may open avenues from a medico-legal standpoint that we do not care or wish to combat. There are too many unproved theories about the thymus for us to have this thrashed out in our courts.

If it were possible to investigate every case of sudden death unexplained by known lesions, especially including those in the first week of life ascribed to atelectasis and congenital heart disease without murmur, every death in infancy due to unexplained convulsions and bronchopneumonia, and every sudden death following the administration of antitoxin, anesthesia, trauma, and minor surgery, the results would be highly illuminating. Although enlargement of the thymus may exist without status lymphaticus or any observed clinical disturbance, nevertheless, hyperplasia of the tissue of the thymus, as a rule, constitutes the key to the general pathological condition. In 118 cases reported by Symmers the thymus was hyperplastic in every instance.

The association of sudden death with thymic enlargement was reported by Morgagni two hundred and fifty years ago, and by Bichat in the Eighteenth Century. Kopp described thymic asthma early in the Nineteenth Century and Lee in 1842 referred to sudden death due to laryngeal spasms and asthma in children. I believe that there is no normal thymus as to size, shape or density, nor is there any constant relation between the thymus and the weight or sex of the child. There is no doubt in my mind that an affection of one side of the thymus might account for some cases of unilateral atelectasis. The mediastinal contents are very movable in infants and young children. It is logical that

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*From the Service, Children's Hospital and Clinic, Tampa, Florida.

there is a definite relationship between the size of the gland and the symptoms manifested. That the symptoms are not due alone to pressure is very strongly suggested by cases in which the upper mediastinal shadow, after reduction by therapy, enlarges to its original size but is unaccompanied by the former symptoms.

Boyd has shown that the fluctuations and the weight of the thymus at any age are concomitant with fluctuations in body weight, and that the growth curve of the thymus in a well nourished child is of the same type as that of the lymphoid tissue in general.

I am going to attempt to demonstrate a few cases to show that the size of the thymus has no relation to the severity of the symptoms. Frequently we attribute a certain train of symptoms to the thymic gland, although in reality the thymus is found negative on post mortem examination. Too frequently sudden deaths in very young infants are classified as thymic deaths, when other factors have caused the symptoms.

Only recently I saw a child who was supposed to have a foreign body in his bronchus. I had seen him on previous occasions; in fact, I had fed him from the age of one month. He was a perfectly well developed, well nourished infant of one year of age. To my knowledge, as well as the parents', this child had on no occasion ever presented one single symptom of a thymic enlargement.

On admission to the hospital for the foreign body examination, this child showed a huge thymic shadow. The foreign body, which happened to be a sand-spur, was removed. X-ray therapy was discussed. So far, this child has yet to present any symptoms which could be attributed to the thymus gland.

We have had several very young infants in whom we felt certain that the thymus might be causing some disturbances. In two of these we were fortunate enough to secure a post mortem examination. One had a congenital heart disease. The other had an atelectasis, with an apparently normal thymus, if we can call any thymus gland normal as to dimensions and weight. I am of the opinion that we have allowed ourselves to become overenthusiastic in blaming the thymus gland for many conditions that we observe in the very young infant. I do not want to go on record as believing that the thymus gland does not cause trouble, but I believe that it causes less trouble than we frequently think. Other respiratory conditions, such as disease of the sinuses and lungs, should be given closer study.

As we learn that many different abnormal conditions can and do occur, we shall certainly become more conservative in prescribing x-ray therapy for every infant that has a cough, laryngeal stridor or other interference with the respiratory apparatus. We have in the past nine years had 25 children treated with x-ray. In reviewing the history of these cases I believe that there are several that would have done just as well had we not instituted x-ray therapy.

It has been demonstrated by different authors, and it certainly has been our experience, that we are too prone to believe that we are dealing with an enlarged thymus gland that is causing obstructive symptoms rather than some other distinctive clinical condition.

Wasson's conclusions are that in the infant under two years of age, the thymus is variable in size, not only in different types of infants, but in the same infant at different times. The underweight infant has a small thymus and the overweight infant a large thymus. The question here rises whether or not the overweight infant should be carefully watched for a large thymus before surgical operations. I do not believe it is necessary in all cases, because we have on several instances done emergency surgical procedures upon such infants who had no untoward symptoms.

For the purpose of description Wasson has classified infants with respiratory stridors in four groups:

(1) The infant, who, at birth, makes a respiratory noise due to mucus.

(2) The infant from one or two months to one year of age who has a stridor varying in character which is usually attributed to the thymus.

(3) The infant with evidence of a tumor or other anomaly to account for the stridor.

(4) The infant with definite and unmistakable evidence of infection of the respiratory passages.

Thus classification places thymic disorders on an equal plane with other respiratory conditions which we are called upon to classify and treat.

I believe that we can and do have a temporary enlargement of the thymus due to intercurrent diseases, such as whooping cough. As Pancost has pointed out, the size of the thymic shadows varies greatly on inspiration and expiration and this should always be taken into consideration in the interpretation of the x-ray plate. The lateral view picture I believe to be far more significant than the antero-posterior view. It has occurred to me that it is possible for thymic enlargement to bear some relation to seasonal or climatic conditions. I am confident that we do not encounter as many enlarged thymus glands as do many of our colleagues in the more rigid cold winters in the North. There is no doubt

that acute respiratory infections will give a temporary enlargement of the gland with accompanying symptoms. When the infections are controlled, the thymic symptoms become less marked, and soon completely disappear. I have, as I said, seen several such cases.

SUMMARY

I believe that we very rarely see a case that needs x-ray treatment. Most cases of thymic enlargement, with accompanying symptoms, are due to respiratory infections. Occasionally we see cases caused by a congenital anomaly. Closer study of the cases by radiologist and pediatrician is needed for us to arrive at definite conclusions. As long as we routinely send all cases of enlarged thymus to the radiologist to be treated we shall remain ignorant of many facts that, by closer scrutiny, we might be able to shed some light upon.

CONCLUSIONS

(1) Do not hasten x-ray therapy in every case of enlarged thymus with respiratory stridor, before a thorough clinical observation for other causes.

(2) Let us consider thymus enlargement not so much as a distinct clinical condition, but as the result of other pathological conditions.

(3) The condition of enlarged thymus demands closer study upon our part with closer cooperation between the radiologist and the pediatrician.

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DISCUSSION (*Abstract*)

Dr. J. D. Love, Jacksonville, Fla.—Owing to our inability to correlate x-ray findings with our conception of thymic disorder, and to our limited knowledge of the size, assumed function and dysfunction of the thymus and what constitutes the true thymic syndrome, one who speaks with too much assurance concerning this subject renders himself subject to adverse criticism, either now or in the near future. While recognizing the value of x-ray findings in the diagnosis of thymic disease, I feel that they should be received merely as contributory evidence in connection with clinical manifestations.

I am inclined to regard as impracticable the suggestion of Dr. Martin of the value of a lateral x-ray picture of the thymus. The technical and anatomical difficulties encountered in securing a distinct lateral picture of the gland almost preclude the possibility of its usefulness.

Possibly, many cases of early thymic enlargement can be explained on the hypothesis that during the early weeks of infancy the suprarenal glands undergo some degree of involution, thus bringing about a resulting enlargement of the thymus. The essayist has pointed out that thymic hypertrophy is a manifestation of complete removal of the suprarenals. In the absence of an accepted thymic syndrome, when we are confronted with a symptom-complex strongly suggestive of thymic disorder, after having eliminated other conditions which might be responsible for the symptoms, and even without confirmatory x-ray findings, I am inclined to the belief that we are justified in employing the x-ray therapeutic test to assist in the diagnosis. On several occasions I have resorted to this procedure with results so satisfactory that I feel justified in its further employment. I advocate this measure only if it is performed by a roentgenologist whose skill and knowledge of proper dosage preclude the possibility of serious injury to contiguous structures, such as the thyroid and parathyroid. Then, even if the x-ray should suppress any assumed function of the thymus, no harm would result, since, as the essayist has pointed out, there are no known ill results following complete thymectomy. On the other hand, should the x-ray therapeutic test stimulate this hypothetical function, the spectacular results following its employment more than justify its use.

Dr. William W. McKibben, Miami, Fla.—Dr. Martin shares the feeling that I have had for some time, that although the thymus is doubtless an important gland of internal secretion, yet our knowledge of its functions is very limited, particularly of its relationship to other glands and their secretions.

Attempts to use the extract of the thymus gland in pluriglandular therapy have seemed to me to be based on insufficient evidence.

It has been brought out in this meeting that there may be an increased area of dullness under the manubrium, the base of the tumor may be felt above the sternum, and there may be an enlarged x-ray shadow; that there may be, too, noisy inspiration and expiration, shortness of breath, retraction, and cyanosis possibly due to pressure not only on the trachea, but on the veins, and nerves; following which insufficient oxygenation of the blood results. The relationship of the noise of the stridor to inspiration or expiration is important in diagnosis.

I have felt that treatment with the roentgen ray is not justifiable unless there are provocative symptoms.

John Morse, of Harvard, like Dr. Martin, emphasizes the fact that, generally, the symptoms attributed to enlarged thymus, may be due instead to congenital heart disease, respiratory infections, atelectasis, cerebral hemorrhage, congenital debility, chilling, adenoids, congenital stridor, laryngismus or stridulus, due to spasmophilia, but that there is no proof that enlarged thymus is the cause of status lymphaticus.

Morse sizes up the situation by saying: "When physicians have the thymus obsession, they overlook the most obvious signs and symptoms and see nothing but the shadow of the thymus gland."

Dr. H. Leslie Moore, Dallas, Texas.—I have no thymic obsession. I have seen many cases of thymic disturbances, and I have seen a few deaths. There are two extremes in this matter. One extreme is the man who is hunting for enlarged thymus and considers every shadow that is even slightly broader than the reputed normal to be an enlargement. That man, of course, is just as far wrong as the man who says there is no such thing and that the thymus is not responsible for any symptoms. One is just as far wrong as the other. There is a medium ground that we should occupy.

A few years ago I ran into a number of cases. They came thick and fast, and they definitely improved under proper treatment. The cases now are not coming nearly so fast; I may be a little more conservative. Some anatomists have found by actual dissection a branch of the vagus going to the nose, which, in all probability, will prove that the pressure of the thymus against the vagus is going to account for the obstructive symptoms. We also have cases of thymus in which there were no obstructive symptoms and yet death occurred, and autopsy proved it to be a thymic death.

Dr. Chas. E. Conrad, Harrisonburg, Va.—I recently saw a child with distinct laryngeal stridor and the history that two other children in the family had had a similar condition, with sudden death in both cases. An x-ray examination showed a definitely enlarged thymus, which responded to x-ray therapy, and the child has done well since. Judging from the history of the two who died, and the definite finding in the one case which I saw, all three children born in this family had thymic enlargement.

Dr. Martin (closing).—Dr. Love considers the lateral view x-ray picture hard to obtain. At present it is difficult to obtain readings of the lateral view, but I believe that after we gain more knowledge in x-ray interpretations this is going to be the position of choice. Because the mediastinal contents are very movable in young children, we must be guarded in our interpretation of whether a thymus is large enough to cause obstructive symptoms. A squirming, twisting, crying infant on an x-ray table at times becomes a major problem in securing a picture that will be of any assistance in interpreting the size of the thymus gland.

Where there is a persistent thymus and the symptoms are slow in disappearing, it is probable that backward pressure is causing the obstructive symptoms that are attributable to the thymus gland.

My plea to you today is that we all approach this subject with an open mind, that we study these cases more closely, and use x-ray therapy only after we are thoroughly convinced that we are dealing with a thymus that needs radiation.

FOOD CONTENT WITH RELATION TO DENSITY AND COMPOSITION OF STONE IN UPPER URINARY TRACT*

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and
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What are the known factors of stone formation?

(1) The precipitation of certain salts that are in the urine.

(2) The part played by the colloids of the urine which maintain the balance of solubility of these salts.

(3) Infection of the urinary passages.

(4) Stasis of the urine.

The indefinite factors of heredity and geographical location are interesting from a statistical standpoint, but too little is understood to form a very definite basis.

In considering the factors separately, we can readily advise the proper steps to diminish or abolish the local infection and suggest the proper remedy to relieve stasis. As to the colloids, we are still almost wholly in the dark and it may be that here eventually will be found the keynote to stone formation, but as to the composition and character of these bodies, all we know about them is that they affect the surface tension of the urine.

That, then, leaves us the question of precipitation of salts. It is quite true that the solubility and excess precipitation of salts is very different in water and in urine. The same physical laws do not govern these two. This is due to the colloid bodies present in urine. The old advice of the physician to prevent reformation of stones by drinking distilled water only is based on the idea of eliminating the salts ordinarily present in water, but the small quantity of these salts taken into the body in solution in ordinary water is infinitesimal in comparison to that ingested with food. The drinking of water is beneficial, of course, in that it increases the amount of solvent. Where the loss of fluids by perspiration and physical work is considerable, the nitrogenous metabolism is high with a correspondingly large output of urea and salts, as R. K. Lee Brown

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has shown. So, that in the matter of diet, water must be definitely considered and its intake prescribed in accordance with the patient's ability to eliminate it.

Supersaturation must exist before crystallization occurs. Foreign material, such as epithelium, mucus, blood, foreign bodies or bacteria, do not cause crystallization in a fluid that is simply saturated. Neither is their presence necessary to start crystallization in a supersaturated urine. The formation of a colloid material which served as a nucleus of a stone was suggested by Rainey, nearly fifty years ago. Such colloid was thought to be the result of desquamative degenerative changes in the renal pelvis or calyces but colloid material of this kind exists in urine under normal conditions. Many salts are held in solution by such colloids. When they are separated from the colloids by dialysis they immediately crystallize. Several hours may be required for a clear alkaline urine to become cloudy, whereas if the colloids be removed crystallization of phosphates occurs rapidly. While it is true that the nucleus of a stone may be a foreign body, blood clot, tissue remnant, parasites or bacteria, it is also true that the majority of stones do not contain such foreign materials but are composed of chemical precipitates and colloids. Renal calculi, then, usually are composed of crystallized inorganic salts, either acid or alkaline in reaction and with a protein framework. Crystallization or precipitation of solids in urine depends partly upon their concentration, partly upon the reaction of the urine and partly upon the presence of colloids. But the presence of this tripod of factors is inadequate to stone formation. There must be stasis. So long as there is a free flow of urine, the crystals will be washed out as "sand"; let obstruction obtain and stone results. Our thesis is, therefore, that there are four factors essential for urinary stone formation and that if any three or fewer be present, stone formation can not occur.

We have classified 29 renal and ureteral calculi according to their density and chemical composition. Our findings are given in the accompanying *Table I*. Summarized, according to constituents, we find:

Calcium present in 85 per cent.
Ammonium present in 62 per cent.
Magnesium present in 51 per cent.
Phosphate present in 51 per cent.
Oxalate present in 24 per cent.
Urates chiefly present in 20 per cent.
Carbonate present in 13 per cent.
Cystin present in 6 per cent.

Thus, with the exception of cysten and carbonates, we have found six salts in over 20 per cent of stones and since these statistics are taken from a distribution too wide to be assigned any local cause and from the average climate of the United States, they may be considered fairly typical. Of this number of examined stones, fifteen were either recurrences or multiple stones, which is over 50 per cent and indicates the importance of further effort to lessen formation.

We had hoped that the relative density of the stone would give us some clue to its composition. One of the standards of radiography is that the specific gravity of a substance depends on its atomic weight. After these stones were removed, we attempted to measure their density, using as a standard aluminum filters in one, two, three and four thicknesses. But we found that those stones that gave the greatest density, that equal to four thickness, outside the body, did not give the greatest density when viewed while in the body. This may be ascribed partly to the size of the stone and partly to the amount and character of tissue of the patient. We can at present see no formula by which the composition of the stone can be arrived at from the x-ray of the stone *in situ*. Practically all of our stones were reported by our chemist as carrying a small quantity of urates and traces of indeterminable organic nitrogenous compounds.

This leaves us to consider, then, the exogenous source of those salts found in removed stones.

Purine Bodies.—Purine bodies are derived largely, perhaps entirely, from the decomposition of nucleoproteins which occur chiefly in the nuclear substance of animal and vegetable cells. The purine bases resulting from the breaking down of nuclei acid are converted into uric acid by two chemical processes. The amino group is removed by deaminization as when guanin is converted into xanthin which is then reduced to uric acid by oxidation. These chemical changes are the result of the action of special ferments present in certain body cells. In the human organism purines are classed as exogenous when they are derived from food and endogenous if they are formed by the decomposition of the nucleoproteins of the tissues of the body.

The uric acid in blood ranges between 1 and 3 mg. per 100 c. c., in normal persons on a mixed diet. It exists in the blood chiefly in the form of monosodium urate though a possible dimorphism is suggested by the work of Morris, Macleod and Benedict. In this connection it is inter-

TABLE 1

Name & Number	Age Sex	Density	Chemical Composition	No. of Stones	Appearance of Shadow Before Removal	Recurrence Remarks
1. J. D. T.	47 M	No. 1	Ammonium urates and cystine	One	Dense	Died
2. L. A. M.	65 F	No. 1 No. 2	Calcium oxalate and ammonium magnesium phos.	Multiple bilateral	Dense	One small stone passed since operation No shadows
3. C. C. S.	23 M	No. 2	Calcium and ammonium magnesium	One	Dense	Nephrotomy No recurrence
4. W. D. M.	50 F	No. 1	Calcium and ammonium magnesium phos.	One	Not dense	Pyelotomy No recurrence
5. S. S.	11 F	No. 1	Calcium and ammonium magnesium phos.	Multiple bilateral	Dense	Died
6. N. D.	42 M	No. 3	Calcium and ammonium magnesium phos. with ammonium urate	One	Not shown	Passed stone following colic, but later many vesical calculi
7. R. J. M.	44 F	No. 1	Calcium and ammonium magnesium phos.	One	Dense	Pyelotomy No recurrence
8. H. T.	22 M	No. 1	Calcium carbonate and triple phosphates	One	Moderately dense	Removed by nephrotomy No recurrence
9. W. L. H.	56 M	No. 1	Calcium phos. and ammonium magnesium phos.	One	Dense	No recurrence Removed cystoscopically
10. H. M.	44 M	No. 4	Calcium oxalate Uric acid Xanthin	Two	Not dense	No recurrence
11. B. M. C.	22 F	No. 1 No. 2	Cystine and ammonium urates	Two	Dense	No recurrence Nephrectomy
12. S. D.	43 F	No. 1	Calcium carbonate Ammonium magnesium phosphate	One	Very dense	Stag horn calculus Nephrectomy
13. S. M. F.	45 F	No. 1	Calcium phosphate and ammonium magnesium phosphates	One	Dense	No recurrence Removed cystoscopically
14. F. D.	28 M	No. 2	Calcium phosphate and ammonium magnesium phos.	One	Dense	No recurrence Removed cystoscopically
15. J. V. StC	60 M	No. 2	Calcium phosphate and ammonium magnesium phosphate	One	Dense	No recurrence Pyelotomy
16. B. S.	30 M	No. 2	Calcium oxalate Uric acid Xanthin	One	Dense	No recurrence Removed cystoscopically
17. W. P.	30 M	No. 2	Calcium carbonate Ammonium magnesium phos.	Multiple bilateral	Dense	Many recurrences Two shadows at present
18. J. C. A.	40 M	No. 1	Calcium phosphate and ammonium	One	Dense	This stone is a recurrence. History of stones 5 years ago
19. R. C.	20 F	No. 3	Calcium oxalate	One	Faint	No recurrence Removed cystoscopically
20. E. H. C.	38 M	No. 4	Calcium carbonate Ammonium magnesium phos.	One	No shadow	Many recurrences. Has passed several small stones
21. C. C.	36 M	No. 3	Calcium oxalate Ammonium urate	One	Faint	This stone is a recurrence of a stone 4 years ago

TABLE 1—Continued

Name & Number	Age Sex	Density	Chemical Composition	No. of Stones	Appearance of Shadow Before Removal	Recurrence Remarks
22. E. O. W.	28 M	No. 4	Too small to report	Multiple bilateral	Faint	Many recurrences Two shadows at present
23. P. M.	21 M	No. 2	Urates—Fibrin Ammonium urates Triple phosphates	One	No shadow	Very small stone No recurrence
24. G. H. B.	40 M	No. 1	Calcium carbonate Ammonium magnesium phosphate	One	Dense	No recurrence Removed cystoscopically
25. W. B. T.	33 M	No. 2	Calcium and Ammonium magnesium phosphate	One	Faint	No recurrence Removed cystoscopically
26. J. L.	40 M	No. 2	Calcium oxalate	One	Faint	No recurrence Removed cystoscopically
27. F. H. K.	35 M	No. 2	Calcium phosphate Ammonium magnesium phos.	One	Faint	No recurrence Removed cystoscopically
28. W. D. P.	39 M	No. 3	Calcium and magnesium phos.	One	Faint	One recurrence Removed cystoscopically
29. A. M.	40 F	No. 1	Calcium phosphate ammonium magnesium phosphate Carbonate and oxalate	Multiple	Dense	No recurrence Nephrectomy

esting to observe that in the increased blood uric acid of gouty patients there occur deposits of urates in certain tissues, while in an equal increase of blood uric acid in patients having leukemia, degenerative liver disease, nephritis, so forth, such uratic deposits do not occur. As an explanation of this peculiarity it has been suggested that monosodium urate occurs in two isomeric forms, one with a solubility up to a concentration of 8 mg. per 100 c. c. of blood, while the other has a solubility no higher than 2.5 mg. It is further explained that the more soluble form is the primary state, which may gradually be converted into the less soluble and more stable salt; that in gout this evolution to the more stable salt occurs, hence uratic deposits; while in leukemia and the other diseases characterized by increased blood urates this evolution does not occur. In either event a supersaturation point of the blood may be reached and in the case of the non-gouty group with the higher gram content per 100 c. c. of blood the point of supersaturation of the urine is more likely to be reached.

The endogenous purine bodies are increased in every condition characterized by increased cell destruction in the body. Such increase of cell destruction is found in pneumonia during the stage of resolution, in all septic conditions, in x-ray exposures, gout, nephritis, malignancy, chronic lead poisoning, arterial hypertension, in leukemia and particularly in acute degenerative processes of the liver. The uric acid concentration in the blood is also high in the child for several days after birth.

The exogenous uric acid in the blood is the contribution of the food the individual eats. Nucleoproteins are present in a number of foods, abundant in some such as liver, sweetbreads, kidney, brain, meat, and scanty in others, such as potatoes, turnips, radishes, and beets. Beverages containing caffeine and similar substances which are substitution products of the purine ring increase the uric acid content since about 15 per cent of caffeine is convertible into uric acid. The idea that white meat contains less nucleoprotein than red meat is erroneous. Fortunately the majority of vegetables have a low nucleoprotein content; this is especially true of those that grow under ground. The legumes, on the other hand, are relatively rich in these substances.

In the care of a patient who has uric acid showers or has had actual uric acid stone formation, diet becomes a matter of some importance. Surgery may remove the initial stone or stones, but the conditions necessary for subsequent reproduction may again obtain. A suitable diet may

TABLE II

LOW PURINE DIET

FOODS ALLOWED:

Bread, White	Cucumbers	Grape Nuts
Fruits	Lettuce	Shredded Wheat
Eggs	Cauliflower	Tomatoes
Milk	Beets	Rice
Cheese (Edam, Swiss, Gervais, Roquefort)	Onions	Tapioca
	Wine	Potatoes
	Nuts	Caviar
	Olive Oil	Butter
	Oatmeal	Sago
Cream		

FOODS IN LIMITED AMOUNTS:

Oysters	Lobster	Cabbage
Crabs	Salmon	Cheese (Cream, American, Cottage)
Hominy	Cod Fish	
Carrots	Radish	
Asparagus	Celery	Cocoa
String Beans	Chicken	Chocolate
Turnips	Brains	Coffee
		Tea

FOODS TO BE AVOIDED:

Broths	Pork	Squab
Beef Tea	Venison	Tongue
Bouillon	Rabbit	Trout
Kidney	Squirrel	Salmon
Liver	Turtle	Perch
Sweetbreads	Beans (Shelled)	Pike
Peas		Herring
Beef	Beans (Lima)	Sardines
Veal	Mushrooms	Anchovy
Mutton	Malted liquors	Goose
Lamb	Lentils	Lobster
	Spinach	Kohlrabi

keep the uric acid content of the urine below the supersaturation point, thus preventing the completion of the circle of factors considered essential for stone formation. A purine free diet is not practicable, but a low purine diet is. Such a diet is illustrated in *Table 2*.

Oxalate of Calcium.—Calcium oxalate crystals may form in either acid, alkaline or neutral urine. From 15 to 20 mg. of oxalic acid are eliminated daily in normal urine, and it is of both exogenous and endogenous origin. The results of studies of the resorption and excretion of oxalic acid derived from foods are, probably, more contradictory than those of any of the crystallizable substances concerned in stone formation. The reaction of the urine appears to have no effect on the solubility or precipitation of oxalic acid. Urine is always supersaturated with calcium oxalate and its solubility appears to be determined by the colloid content. The excretion of oxalic acid, apparently, bears no relation to protein metabolism, although in the test tube uric acid can easily be changed to oxalic acid. There seems to be an interdependence between nucleoprotein metabolism and oxalic acid formation. Its excretion is increased by the ingestion of gelatin. Since gelatin contains glycocoll it was thought that other foods, such as fowl, which contain glycocoll, served as oxalic acid sources, but feeding glycocoll causes no increase in oxalic acid output. Creatin is transformed into oxalic acid and since it occurs in muscle of all vertebrates, especially fowl, this is probably one of its sources. Foods that contain oxalic acid already formed constitute an unquestioned source of urine oxalate; that some is formed from foods not already containing it, as either the free acid or a salt, seems established, but the chemical metamorphosis is not understood; that some is formed as the result of metabolism of the body is also known, though the details are not yet available. We know only that oxalic acid is a normal body metabolism product. The larger part of oxalic acid found in vegetables occurs as an insoluble calcium salt which is favorably affected by the hydrochloric acid of the stomach. In fact, oxalic acid excretion by the kidney can be increased by the administration of hydrochloric acid and diminished by giving alkalis.

In the effort to prevent reformation of oxalic acid stone it must be borne in mind that a decreased calcium and an increased magnesium content with a highly acid urine are desirable conditions. The diet should be low in carbohydrates since their fermentation favors both the formation and absorption of oxalic acid. Foods containing a high oxalate content should be restricted or eliminated. Such a scheme is indicated in *Table 3*. In arranging such a diet three desiderata were kept in mind: first, exclusion of foods with a high oxalic acid content; second, avoidance of foods which contain less magnesium than calcium; and, third, the inclusion of foods which increase the acidity of the urine, thus increasing its solvent power for oxalates.

PHOSPHATES

Since one of the functions of the kidney is that of assisting in the maintenance of body neutrality by variances in the excretion of phosphates and ammonia, some reference to hydrogen ion concentration of the blood becomes necessary.

The older terms acidity and alkalinity in relation to body fluids are now recognized as being only approximate and relative. Biochemistry set up a new order by introducing the ionic conception which gave to the term reaction a quantita-

TABLE III

DIET FOR OXALURIA AND OXALATE OF CALCIUM CALCULI

FOODS ALLOWED:

Fruits:

Apples	Peaches	Pears
Apricots	Melons	Pineapple

Vegetables:

Lima Beans	Potatoes	Artichoke
Shelled Beans	Lentils	Yams
String Beans	Rice	Frijoles
Peas		

Breads:

Wheat	Rye	Buckwheat
Corn		

Cereals:

Oatmeal

Meats:

Beef	Fish	Chicken
Veal	Oysters	Duck
Mutton	Rabbit	Turkey
Lamb	Squirrel	Goose
Pork	Squab	

Fats:

Vegetable:

Olive Oil	Cotton Seed Oil	Nuts

Animal:

Butter	Cream	Lard
Cheese	Suet	Bacon

Beverages:

Water:

Plain	Carbonated
Whisky	Brandy

FOODS ALLOWED IN LIMITED QUANTITIES:

Fruits:

Bananas	Grapefruit	Raspberries
Cherries	Lemons	Blackberries
Avocado	Oranges	

Vegetables:

Lettuce	Rutabagas	Brussel Sprouts
Onions	Cucumbers	Okra
Parsnips	Cress	Pumpkin
Brocoli	Egg Plant	Squash
Leeks		

Milk:

Condiments

FOODS TO BE AVOIDED:

Fruits:

Cranberries	Currants	Figs
Strawberries	Grapes	Dates
Gooseberries	Plums	Olives

Vegetables:

Rhubarb	Carrots	Cabbage
Sorrel	Radishes	Cauliflower
Spinach	Peppers	Turnips
Celery	Asparagus	Endive
Tomatoes		

Meats:

Liver	Gelatin	Calves' Foot
Sausage	Sweetbread	Jelly

Eggs:

Yolk	Shad Roe	Caviar

Beverages:

Tea	Cocoa	Chocolate
Coffee		

tive meaning indicating the degree of alkalinity or acidity existing in any solution. The chemical and physiological properties of such a solution are determined, largely by the degree of ion content. Dissociable substances, such as alkalies, acids, and salts separate, in solution, into components called ions. Alkalinity and acidity become, then, a matter of whether much or little hydrogen ion is present.

The body fluids in health are existing under conditions which tend to change their reaction or hydrogen ion concentration. The blood is the medium through which the various parts of the body are kept at the same hydrogen ion concentration. The normal hydrogen ion concentration of the blood is equivalent to one gram of hydrogen in 32 million litres of water, which puts it just on the alkaline side of reaction.

As a matter of convenience, Sorensen suggested the sign cH for actual hydrogen ion concentration and expressed it numerically as a power of ten. The phraseology was further simplified by using only the numerical value of that power and this index is designated its pH. Neutrality is expressed as pH 7.0, acidity as varying between pH 0.0 to 7.0, while alkaline solutions range from 7.0 to 14.0. Since pH represents a negative logarithm it follows that as pH increases the acidity decreases.

Expressed by this nomenclature the reaction of the blood is pH 7.4 in health and the range of variation from this is extremely small, though life is said to be possible between the ranges of pH 7.2 and 9.0. There are five factors engaged in preserving the normal limits of blood pH: viz: blood salts, kidneys, liver, lungs and intestines. The relationship of the first two only is to be considered here.

The pH of the average urine is 5.5 to 7.0. In other words, normal urine is acid. The fairly constant pH of the blood is made possible because of the presence of "buffer" salts designed to take up excess hydrogen ions to be excreted by the lungs and kidneys in less active forms. These buffer salts are sodium bicarbonate and alkaline sodium phosphate. Considering only the alkaline sodium phosphate, it will be recalled that the chief store of this salt is in the red blood cells. When an acid is added to the blood, as

from foods, the alkaline phosphate is transformed to an acid phosphate and alkali is thus released, while the acid phosphate is eliminated by the kidney. This acid sodium phosphate is the chief acid constituent of the urine and determines its reaction. If, however, there should occur an unusual acid loss, as in over-stimulation of the respiratory center, no acid phosphate or much less than the normal amount is eliminated in the urine and it becomes alkaline because the excess phosphate is eliminated as an alkaline salt. When the available alkali of the blood is reduced by its combination with an excess acid the carbon dioxide combining power of the blood becomes correspondingly lowered. It is this condition which is spoken of, clinically, as acidosis, but which is, in actuality, only a reduction of the alkaline reserve of the blood.

With these facts as a fundamental background, in order fully to understand phosphaturia or phosphatic stones, it is next necessary to study the metabolism of foods containing phosphorus. Food phosphorus is exhibited as the salts of magnesium, potassium, sodium and calcium, and occurs in nucleoproteins, phosphoproteins and in other proteins. While phosphates may be precipitated in a neutral or even a faintly acid urine, phosphatic stones probably occur only in an alkaline urine. Such a urinary reaction may result from vegetable diet, from the ingestion of alkalis, or it may obtain as a result of abnormal conditions in the body.

But we do not believe that the cloudy urine consequent to ammonia changes in an infected urinary tract should be called a phosphaturia; neither should precipitation of phosphates in urine made alkaline by the enormous loss of acid which occurs in persistent emesis be reckoned as phosphaturia. The origin of phosphaturia is outside the urinary tract. The changes in urine during a session of phosphaturia surely are not the result of a mere increase in the excretion of phosphoric acid. It seems, rather, to depend upon an acid elimination disturbance, plus an alkaline state of conservation. Disturbances of the nervous system seem closely linked to phosphaturia and if the activity of the kidney be influenced by the nervous system as appears true, then Minkowski may be correct in recognizing phosphaturia as a secretion neurosis of the kidney.

On this basis, food plays a secondary role in the correction of the condition, a role confined to the determination of the alkalinity of the urine since the separation of a phosphatic sediment should be considered as an indication of lessened acidity. The diet should consist of such foods as tend to increase the urine acidity, thus avoiding precipitation, which is one of the factors in stone formation, or in the growth of a stone already formed. Foods containing calcium should be reduced to bare body needs, thus keeping the phosphate in a more soluble form.

If hyperchlorhydria exists the diet should also encompass the effort to lessen its formation. A concentrated dry diet increases the acidity of the urine as do nitrogenous vegetable and animal foods. The idea that high urine acidity is desirable to prevent phosphatic stone formation should not, however, cause one to lose sight of the danger of an extreme in this direction, which might create a precipitation of solids of an acid nature. It is in this fashion that the so-called laminated stones are formed, the nucleus of the stone being phosphates, while the subsequent layer may be uric acid.

TABLE IV

DIET IN PHOSPHATURIA

FOODS ALLOWED:		
Corn	Beef	Squirrel
Bread	Fowl	Oatmeal
Eggs (white)	Pork	Peanuts
Fish	Rabbit	Rice
Butter	Cream	Olive Oil

FOODS ALLOWED IN LIMITED QUANTITIES:		
Bread (wheat)	Onions	Milk (skimmed)
Eggs (yolk)	Turnips	Rutabagas
Meats	Radishes	Spinach
Cocoa	Oysters	Tomatoes
Mustard	Salmon	Turnips
Asparagus	Cocoanut	Gooseberries
Beans (string)	Cucumbers	Huckleberries
Brussel Sprouts	Egg Plant	Strawberries
Cauliflower	Lentils	Currants
Celery	Lettuce	Pears
Clams	Parsnips	Apples
Corn	Peas	Apricots
Chestnuts	Peppers	Oranges
Artichokes	Pumpkin	Cherries
Potatoes	Rhubarb	Figs
Cabbage		

FOODS TO BE AVOIDED:		
Beans:	Beets	Prunes
Lima	Carrots	Raisins
Shelled	Almonds	

A suggested diet is presented in *Table 4.*

DISCUSSION *(Abstract)*

Dr. A. J. Crowell, Charlotte, N. C.—I know nothing about the relation of food content to the opacity and composition of upper urinary tract calculi with the exception of that produced by cystin.

I had hoped that Drs. Grant and Simpson would give us something more definite in the way of preventing the formation of nephrolithiasis and that possibly they might give us something to bring about disintegration, but lo and behold, my hopes have not been realized!

I believe, however, there is a relationship between the intake of food and nephrolithiasis. I am quite positive in my mind that there is a relationship, insofar as the etiology is concerned, between infection of the kidney and stone formation.

It has been very clearly demonstrated that there is a definite relationship between faulty metabolism and cystin nephrolithiasis. Possibly we have more of it than we think. We have found in our clinic only seven cases of this disease. We were fortunate to keep one of these under observation sufficiently long to bring about disintegration of three stones in the left kidney and a large one in the right, varying in size from 5 to 20 mm. in diameter. This patient had been operated upon about six and three months before, when large stones had been removed from each kidney. A rapid recurrence took place, with infection in both kidneys. By restricting the protein intake, keeping the urine strongly alkaline, and lavaging the kidney pelvis with hot alkaline solutions, we were able to bring about disintegration of these stones within nine months. The patient remained free from stone, insofar as we could ascertain while she lived, which was between two and three years after their disintegration, when she died of pneumonia. This naturally leads me to hope that some of these days we shall find some way of preventing the formation of other forms of kidney stone.

Pelvic lavage should be used following pyelotomy, certainly in cases of infection, to clear up the infection and get rid of any debris that may be left in the kidney. The patient is usually anxious to get home after the operation and I fear we are not so persistent as we should be in carrying out this postoperative treatment. The presence of any debris in the kidney acts as a nucleus upon which the salts of the urine may deposit.

Dr. Neil S. Moore, St. Louis, Mo.—Osborn and Mendell sometime ago accidentally discovered that rats fed on a diet deficient in vitamin A developed phosphatic kidney stones. They admitted that such a deficient diet did permit a debilitated condition in rats; and McCullom thought that was the reason for the formation of the stones. Later, the work of Osborn and Mendell was confirmed by two independent workers and they were able to produce phosphatic stones in the kidney in forty and thirteen days, respectively.

I have attempted to classify roughly 150 cases of urinary calculi. They were private patients of practically all nationalities, most of them from St. Louis. The diet that charity clinic patients eat has not a great deal to do with stone formation.

From our series of cases I do not think heredity is an important factor. Geographical location is more important. We find a great number of stones in Italian and French patients, and it is generally known that stone is rather rare among negroes. Perhaps the large quantities of wine consumed by most French and Italians may have some bearing on the formation of stone.

Whether or not the stone shows in the skiagraph, depends upon several factors, namely: preparation of the patient and elimination of gas in the gastro-intestinal tract, the size and location of the stone, and its consistency. In many cases where the stone is in front of the pelvic bone it does not show well, because its density is equal to that of bone. Of course, the wax bulb catheter, complete obstruction to any catheter and ureterograms are of great aid. Occasionally it is necessary to depend on deposits of silver or iodide salts around the stone to show it. About 5 per cent of our cases were of uric acid or cystin composition and did not show in any situation.

Possibly all of us feel that stones are the result of faulty metabolism, whatever may be the cause for it.

Stasis should never be overlooked. After we have removed the stones we usually incise the ureter opening with our particular ureter opening incisor, which I presented in 1926, the so-called fish fin incisor, later passing instruments to relieve strictures where it is practicable and possible. Finally, the old reliable remedy, of large quantities of water by mouth, is of value in diluting the urine and washing away the average nucleus that may be the cause of future stones.

Dr. M. M. Coplan, Miami, Fla.—We believe that faulty metabolism of particular foods is the underlying cause of stone. In starting a food study some three or four years ago, we wrote to ninety hospitals throughout the country, asking them to give us the number of cases of urolithiasis that had been turned over to the urological service, omitting venereal cases. Only about thirty answers were sufficiently accurate to be included here. We then obtained from ninety city laboratories, where the hospitals were, water analyses for a certain day. Stones are very common in Florida, particularly, however, among the newcomers. In Key West, which has no artificial water supply, but uses all rain water, urolithiasis is common.

In a long list of cases of stone we had only one in a negro, and he was at best a mulatto. Of thirty physicians to whom we wrote in the southern section of Georgia, none had seen a stone in a negro. Dr. Holmes has presented a paper covering this work to the *American Urological Journal*.

Dr. J. Ullman Reaves, Mobile, Ala.—My attention was called some years ago to the chemistry of urinary calculi by Dr. Crowell, of Charlotte. In checking the urine microscopically for evidence of stone crystals those of cystin are readily found in a case where the patient is a cystinuric. I reported a case in New Orleans, and showed three lantern slides; the first, a large staghorn calculus in the kidney; the second was a pyleogram showing this stone to be entirely within the kidney pelvis; the third showed a complete absence of shadow-producing substance in the region of the kidney. This patient's urine was full of cystin crystals and the disappearance of the stone was accomplished in three weeks' time by alkalinization of the patient internally and lavaging the kidney pelvis with an alkaline solution cystoscopically. By carrying out my diet instructions the patient has had no further formation of cystin stones. In reporting this case I made the statement that when we understood the chemistry of metabolism the treatment of urinary calculi would be medical rather than surgical. I am today of the same opinion.

SOME COMPLICATIONS OF OTITIC SEPSIS, WITH CASE REPORTS*

By Sam B. Marks, M.D., F.A.C.S.,
Lexington, Ky.

It is the writer's purpose to give a brief analysis of the mastoid cases occurring during 1928 and 1929 in the practice of his associate, Dr. W. N. Offutt, and himself and not to discuss the many complications of otitis media and mastoiditis nor to compile a bibliography upon the subject. Those desiring such information should refer to Kopetzky's[1] "Otitic Surgery," recently revised, and the many articles by the same author, Braun's[2] "Sinus Thrombosis," Eagleston's[3] "Brain Abscess" and recent articles, Campbell's[4] recent review upon "Complications of Otitis Media and Mastoiditis" and to the work of Kolmer[5] and his associates, entitled "The Chemo-Therapy and Serum Therapy of Pneumococcic and Streptococcic Meningitis," all of which carry full references.

There were performed by us during the year 1928 only eight operations for acute mastoiditis, three of which were of the hemorrhagic type, all of which were further complicated: one with sinus thrombosis; one with serous meningitis; one had a double operation following hemorrhagic measles, and later developed a septic pneumonia, empyema and three metastic abscesses; and one had a rather profound toxemia. All, with the exception of the case of serous meningitis, presented blood cultures positive for the *Streptococcus hemolyticus* and all recovered.

The only other case presenting complications was a neglected otitis media and mastoiditis in a woman of sixty-five, with an extensive sinus thrombosis, who died three days after operation from what appeared to be septic intoxication with heart involvement. These were all cases of the writer's.

During the first six months of 1929, sixteen operations were done for acute mastoiditis, five of which were double and five of which were of the hemorrhagic type, four of these latter being further complicated: one with a large perisinus abscess, who recovered; one with an acute exacerbation superimposed upon a chronic middle ear disease of long standing with a streptococcic systemic invasion, perisinus and extradural abscesses, sinus thrombosis, acute nephritis and postoperative septic pneumonia, who died upon the fifteenth day; and two with septic meningitis following double operations with sinus phlebitis without thrombosis, which are to be reported in detail.

Complications in the group, all of whom recovered, were: one, a case of Dr. Offutt's, a double operation with an acute nephritis; one with facial paralysis; and one in a syphilitic woman thirty-five years old, who had developed an extensive sinus thrombosis. A third operation was necessary in her case for the excision of the posterior one-half of the horizontal portion of the sinus wall, which had previously been evacuated only of the clot, before the blood culture became negative and the temperature excursions ceased.

Case I.—A white boy, aged seven years, presented the following history: in May, 1928, he was seen by the writer during an attack of influenza. A double acute otitis media developed which subsided in a few days with complete healing, after a spontaneous rupture on the left and a right myringotomy. Tonsillectomy and adenoidectomy were advised at this time, but the advice was disregarded.

He was seen by my associate, Dr. Offutt, on January 9, with a right otitis media which healed promptly after myringotomy. The boy was not ill and attended school, but the ear re-opened and closed two or three times. On January 27, Dr. Offutt again saw him with Dr. T. M. Marks in an attack of influenza and a severe double otitis media, for which a double myringotomy was done under chloroform. As his temperature continued high with ears freely discharging thin pus, the left membrane, which had begun to heal, was re-opened four days later and the right, for the same reason, was re-opened in two days. After each re-incision his temperature dropped, but soon was up again from 102 to 103° each day. After being seen by Dr. Offutt, Dr. Marks, and the writer he was removed to the Good Samaritan Hospital on the afternoon of February 1. At this time his temperature was 98°, pulse 110 and respirations were 24. Both ears were open and freely discharging thin pus with slight edema of the postero-superior canal walls, but there was no tenderness nor swelling over the mastoids. The urine was normal except for a trace of albumin, hemoglobin 56 per cent, red blood cells 3,150,000, white blood cells 9,700 with 75 per cent polymorphonuclears and no eosinophils. An x-ray made upon admission was reported negative by the roentgenologist, but after careful study slight feathering about the antrum extending posterior to the canal to the tip was demonstrated on each side. Pus and granulation in those areas, without cell destruction, were then diagnosed. Density was more evident upon the right side. The next day his temperature made a sudden rise from normal at eight a. m. to 105° at four p. m. and immediate operation was done by Dr. Offutt. General examination at this time was negative.

Both mastoids were hemorrhagic with pus and granulations in the antra and in the deeper cells behind the canal to the tip. The tip cells and other cells, including those over the sinuses, contained only thickened mem-

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brane. Both sinus plates were intact and clean with little or no involvement of the zygomatic roots. The right side showed the greatest involvement. Pus from the mastoid showed *Streptococcus hemolyticus*. Blood for culture taken at this time proved to be negative.

The first day showed nothing ususual except a rise of temperature from normal to 104° from eight p. m. to eight a. m. The same thing occurred on the next day and 300 c. c. of whole blood were given by Dr. Francis Massie. A severe chill followed and a temperature rise from 100 to 104°, with hemoglobin 60 per cent, red cells 3,300,000, white cells 16,900 and 81 per cent polymorphonuclears. No malarial organisms were found and the Widal test was negative. General examination was also negative. One-half c. c. of streptococcus immunigen was given subcutaneously and repeated daily for four days, without effect. The next, the fourth day, he developed redness, soreness and tenderness of the left great toe joint, which subsided in three days. Since his temperature had shown no subsidence and his condition no improvement, a Tobey-Ayres test was done by Dr. T. M. Marks on the seventh day, showing a pressure of 150 mm., which readily rose to 300 upon pressure upon either internal jugular, so the test was declared negative. The spinal fluid was clear, contained 5 cells per mm., gave a normal colloidal gold test and no growth upon culture. In the absence of neurological signs, with normal ocular muscles, no nystagmus, normal fundi and negative general examination the expectant treatment was resumed. The next, the eighth day, his blood count showed hemoglobin 56 per cent, red cells 3,510,000, white cells 12,150, 68 per cent polymorphonuclears, and 1 per cent eosinophils. Blood cultures were still negative. Three hundred and fifty c. c. of whole blood were given with no apparent reaction. After a consultation with Dr. Stucky we decided to call Dr. Sam Iglauer, of Cincinnati, who saw the patient that night, and all agreed upon an investigation of the lateral sinuses, which was done the next morning by Dr. Iglauer.

The right lateral sinus, upon exposure, showed one small spot of change in color of the wall suggesting inflammatory invasion. The left was exposed but was found normal. The right internal jugular was then ligated and divided above the facial vein and the neck wound closed with tissue drain. A specimen of blood for culture was taken from the sinus which later proved negative. The sinus was opened and explored, but no clot was found. The sinus had to be packed without excision because of severe hemorrhage, probably from the superior petrosal sinus.

For the next two days he was much better, brighter, less fretful, took more nourishment and slept well, his temperature rising only to 102° each day, but it was 104° at eight p. m. the following day. Fearing an infected clot, we removed all the packing, but no clot was found, free bleeding being immediate. The next, the fifth day after the second operation, was uneventful until midnight, when his temperature in six hours had risen from 101 to 105°. At the suggestion of Dr. Iglauer the next morning, all packing was taken out, the sinus wall excised and free bleeding again established, but to no avail, as his temperature remained up between 103 and 104° and he was increasingly restless.

An aphasia for certain words appeared in the early afternoon of the same day with a slight ankle clonus on the left side, but no other neurological signs. Spinal puncture revealed a cloudy fluid with 130 mm. pressure and containing *Streptococcus hemolyticus*. Twelve c. c. of antistreptococcus serum were given after removal of 30 c. c. of fluid. Both fundi showed slight distention of the retinal veins. A right sided localized meningitis was diagnosed (the boy being left handed). Dr. Iglauer was consulted, who advised taking him to Cincinnati on an early train the next morning. After consulting with Dr. B. N. Carter, a neurological surgeon, and Dr. L. A. Lurie, a neurologist, we agreed upon the diagnosis and an exploration of the right hemisphere was decided upon.

Operation by Dr. Iglauer.—The dura of Trautman's triangle was exposed and found normal. The dura over the tempero-sphenoidal and lateral lobes was then uncovered for an area of about 8 cm., a large localized collection of thin fluid and thick pus of about three ounces in amount was evacuated, rubber drains were inserted beneath the dura and a vaseline rubber tissue dressing put on, which was frequently changed and kept wet. Complete aphasia resulted, a left sided hemiplegia developed and in spite of supportive measures and frequent spinal taps, death resulted in six days. Autopsy was not obtained.

Case II.—A white boy, age five years, a patient of the writer's, had had both ears opened for a simple otitis three years previously. The condition healed in ten days, at which time the tonsils and adenoids were removed. The left ear was opened two years later and again in January of this year, both times for a simple otitis which healed in a few days.

The writer again saw the patient at his home in an adjoining town March 9, with an acute upper respiratory infection and suspected scarlet fever. His mother was ill with severe tonsillitis and his father was just recovering from tonsillitis and quinzy. No history of a rash from either their physician or the family could be obtained, but both the patient and mother desquamated a few days later.

Examination revealed a discharging left ear of five weeks' duration and an acute suppurative otitis with intact membrane on the right side. Both membranes were widely opened under chloroform and directions were given for irrigations with 2 per cent mercurochrome and 5 per cent neo-silvol instillations in the nose. His temperature was 102°.

Three nights later he was seen by Dr. T. M. Marks, who found him still quite sick with a small spot of superficial edema above and in front of the right auricle, and had him brought to the Good Samaritan Hospital the next morning. Both ears were discharging freely a thin muco-pus with large free openings, with no thickening in the membrane nor canal walls. The swelling above the right ear was present, but was only slightly tender and not red. Ice was used for it and it soon resolved. No mastoid tenderness was elicited. General examination was negative with one plus albumin in the urine, hemoglobin 73 per cent, red cells 3,500,000, white cells 13,800 and polymorphonuclears 71 per cent. His temperature was 99° on admission and the highest for the day was 101° by rectum. X-ray of the mastoid was reported negative, but the pictures were not clear. The next day his temperature reached 103° (all temperatures reported are rectal). The next day his temperature was 104° at four p. m., 102° at eight a. m. and 105° at noon. He was re-x-rayed by Dr. D. B. Harding, of the Lexington Clinic, who reported increased density over the cellular area extending from the antrum to the tip, with no destruction of the cells. The right side showed much less change, but slight density of

the same region was evident. The leucocytes had risen to 20,000 with 74 per cent polymorphonuclears. A double mastoid operation was done the same afternoon. Both mastoids were hemorrhagic and there was thin pus and thickened membrane in the antrum, tip and deep cells and extending into the zygoma, involving only two or three cells. The cells over the sinus in each side contained only a thickened membrane, with no pus and no signs of sinus invasion. Culture taken from the mastoid at operation showed *staphyloccus aureus,* which was not the prominent offender, as later events proved. A blood culture made on the day of operation was reported negative.

For the next two days his condition was good, but he looked toxic. The second day his temperature again was 105°, hemoglobin 50 per cent, red cells 3,480,000 (cells small and pale), white cells 18,700, and 89 per cent polymorphonuclears. The third day showed no change with temperature ranging from 101.6 to 104.8°. The neurological findings were negative. A spinal puncture done under chloroform showed a negative fluid with normal pressure. The Tobey-Ayres test, as was expected, revealed no difference in the two sides, but was of course not relied upon. The eye grounds were negative. On the fourth day he was the same and 300 c. c. of citrated blood were given by Dr. Massie, followed by no reaction. Both wounds were in normal condition. Very little food except fruit juice was taken and he had vomited once before transfusion. The urine showed no acetone. The fifth day showed no change except an 8 per cent increase in hemoglobin and 25 per cent polymorphonuclear reduction. The sixth day showed a positive blood culture taken forty-eight hours previously, a temperature from 99 to 104.4° at four to six hour intervals with a slight chill at one rise. The left disc showed moderate papilledema and the right some engorgement of the veins. A moderate Kernig was evident on the left, but no other signs of irritation were present. The Tobey-Ayres test showed 120 mm. pressure and, while not positive, showed a less active rise and fall when the right jugular was compressed. Dr. J. A. Stucky saw him with us and agreed that the right sinus should be investigated.

Operation revealed a full pulsating sinus with a small mural clot upon the vertical wall. The internal jugular was ligated and divided and the sinus opened under compression above and below. After removal of the clot it had to be packed without excision because of severe bleeding. Three hours later he had a temperature of 103.8°, but with no other upset. The following day was a better one with temperature ranging from 101 to 103° in a sixteen hour interval. The third day in the morning he took more food and water, and his temperature was not above 100.4° all day. A second transfusion of 250 c. c. citrated blood was given by Dr. Massie, followed by a sharp reaction with chill and temperature of 105.6°. The fourth day showed no change, with a temperature rise to 103.2° and the wounds in satisfactory condition. The fifth day showed one rise of temperature to 104.6°, with 61 per cent hemoglobin, 3,520,000 red cells with slight cell changes, 17,500 white cells and 81 per cent polymorphonuclears. The right fundus was normal and the left much improved. He was taken to the operating room, given chloroform and all of the outer sinus wall was removed. Free bleeding was elicited and the plugs replaced. The sixth day showed a positive Kernig on the right with a drooping left lower eyelid which he could not quite close. The spinal fluid was clear, showing 36 cells, but no bacteria were found after microscopic examination and culture.

After another consultation all agreed that it was best to investigate the left sinus. This was done immediately. A full pulsating sinus was revealed with a moderately thickened and discolored wall, but no mural clot could be felt. It was covered with vaseline gauze and not opened. After forty-eight hours the wall appeared normal. The next morning at two o'clock, the twelfth day since mastoid operation and the seventh since the right sinus operation, he had a slight convulsion of five minutes' duration and became very nervous, but was quiet after having one-eighth grain of codeine. At nine a. m. contraction of the left hand and arm were evident, which soon showed in the leg and ankle at intervals. There was no definite paralysis, as he could move both limbs freely, but he could not make a coordinate effort. The left disc was now choked and the right slightly edematous. There was a marked Kernig and ankle clonus on the left side and beginning rigidity of the neck, but no nystagmus. Spinal puncture revealed a highly increased pressure and 40 c. c. of cloudy fluid were withdrawn which contained 1,285 cells and streptococci both on smear and culture. Dr. B. N. Carter, of Cincinnati, saw him upon the afternoon of this day, but did not believe that anything could be done for him. After the puncture his temperature dropped gradually from 105.2 to 102.4° in eighteen hours, but was 104° at midnight, going to 103.8° at eight the next morning, when another spinal puncture revealed the same fluid with 3,500 cells and many streptococci. The left arm and leg showed irritation and by noon were paralyzed and he became somnolent, but was easily aroused, took fluids frequently and asked for ice cream. Codeine, one-eighth grain, was given as required to control the persistent twitching in the left arm and leg. The next twenty-four hours showed some general improvement in all respects. He could move the left hand, but the twitching continued with intensity. There was a general convulsion at six p. m. The next morning, the fifth day of the meningitis, he had a general convulsion at one o'clock lasting an hour, after which he began to urinate involuntarily. Much less water was taken, so 1,000 c. c. of saline were given by hyperdermoclysis. Spinal puncture revealed a much less cloudy fluid with little pressure and only 900 cells, with only 1 per cent leucocytes and 2 per cent epithelial cells, no streptococci being found in smear; but there was little change in the neurological symptoms. Another 1,000 c. c. of saline were given. Dr. Carter was again consulted and advised waiting twenty-four hours for culture reports on spinal fluid and general progress.

The following morning at eight, though his condition was worse, with a temperature of 102° and a negative culture of the spinal fluid, we felt justified in be-

lieving the meningitis had fairly definitely localized and Dr. Carter came again and explored the right hemisphere through a small subtemporal decompression, finding a localized collection beneath the dura of about two ounces of fairly thick pus. This he evacuated and drained with rubber tissue. A spinal puncture done on the table revealed a much more cloudy fluid with 7,000 cells. The meningitis had probably become generalized during the morning. Death occurred forty hours later. No autopsy was obtained.

CONCLUSIONS

These two cases illustrate the progressive industry of the *Streptococcus hemolyticus*, and to those who witnessed the cranial operations, the futility of any of the procedures now practised to arrest this type of meningitis, unless studies such as those being pursued by Dr. Kolmer are proven of use.

The unusual picture of a diffuse meningitis tending to localize, which occurred in the second case, was startling to us. None of us had seen it before and all were convinced that if there was cistern blockage it was only partial.

It is the writer's belief that all streptococcic otitis cases must be regarded as potential meningitis as well as potential mastoid disease and that surgical intervention, in any case of streptococcic otitis with persistent fever and evident toxemia, must be instituted very early if one hopes to avoid serious catastrophe.

The careful study of well made x-ray pictures taken early is of great value and cannot be too greatly emphasized, as of diagnostic, as well as prognostic value.

And finally the writer would indeed be very remiss if he failed to take this opportunity of expressing to those associated with him in the conduct of these cases his appreciation and gratitude for service generously rendered.

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Discussion follows paper of Dr. Tunstall, page 645.

THROMBOSIS OF THE SIGMOID SINUS AND INTERNAL JUGULAR VEIN WITH SPECIAL REFERENCE TO THE TOBEY-AYER TEST*

By CUTHBERT TUNSTALL, M.D.,
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Having occasion, last spring, to read up on the subject of sinus and jugular thrombosis, I came across an observation reported by Kopetsky[1] contrary to the results of Tobey and Ayer.[2] Then I found that Kopetsky[3] had elsewhere spoken highly of the Tobey-Ayer test. This had, I felt, been of such great assistance to me in two recent cases, that I undertook to determine, so far as possible with the limited facilities at my command, how much reliance can be placed upon the information furnished by it.

I am indebted to Dr. D. C. Smith, of the Dermatological and Syphilogical Department of the University Hospital, for the manometric readings in a series of routine lumbar punctures and to Dr. F. D. Woodward, of the Department of Otolaryngology, for permission to report one of his cases in which I was privileged to assist him.

In regard to the normal findings in the Tobey-Ayer manometric test, I have prepared charts showing graphically the results of routine lumbar punctures done on patients with no history indicating any sinus or jugular thrombosis. Most of them were luetic. In the charts the columns are arranged in groups of four. In each group of four the first column (1) shows the initial pressure of the cerebrospinal fluid; the second column (2) shows the pressure when the right internal jugular vein was compressed; the third column (3) indicates the pressure when the left jugular vein was compressed; and the fourth column (4) when both jugulars were compressed.

Tobey and Ayer,[2] in their original communication on the subject, and Tobey[4] a few months later, called attention to four pitfalls to which the test is liable. These are:

(1) An absence of rise or very slight rise in patients with very low cerebrospinal fluid pressure.

(2) Faulty technic causing incomplete compression of the jugular veins. This is especially likely to occur in patients with thick necks due to obesity, or tumors.

(3) An abnormal rise in pressure due to straining

*Read in Section on Ophthalmology and Otolaryngology, Southern Medical Association, Twenty-Third Annual Meeting, Miami, Florida, November 19-22, 1929.

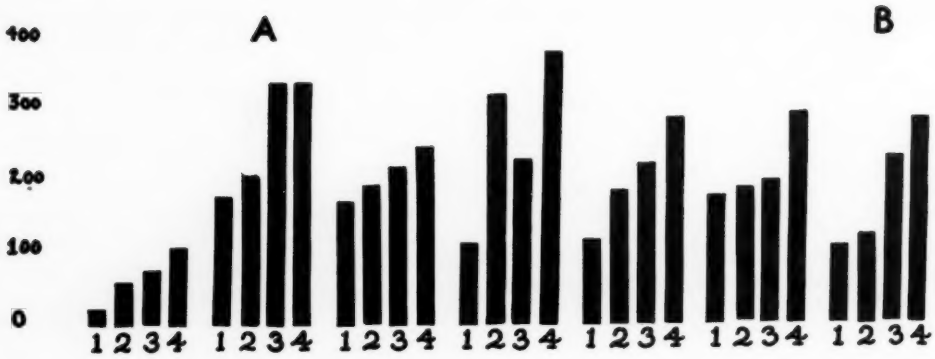

Chart 1

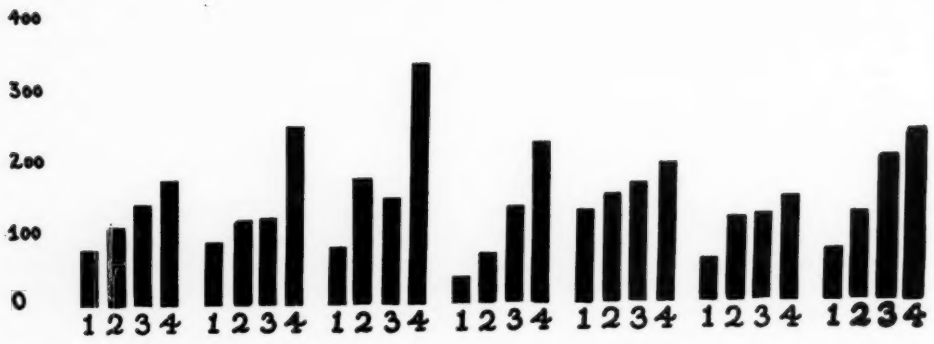

Chart 2

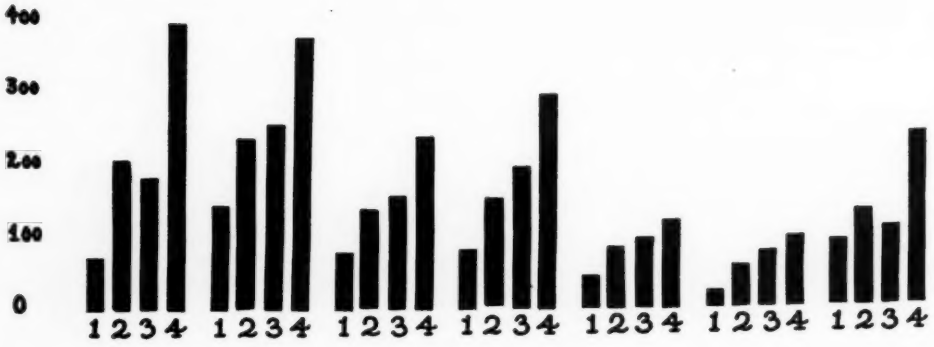

Chart 3

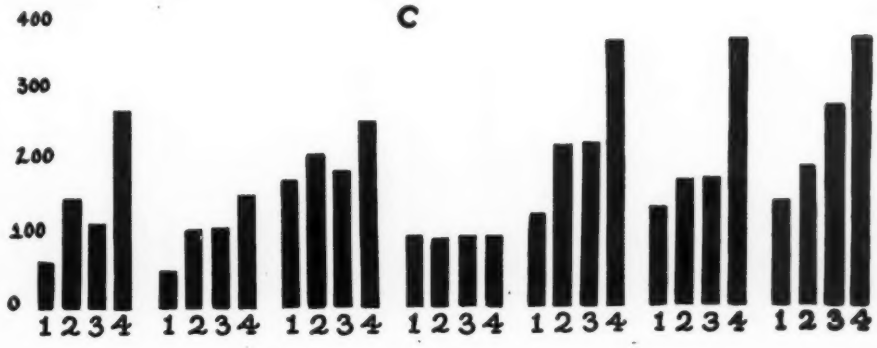

Chart 4

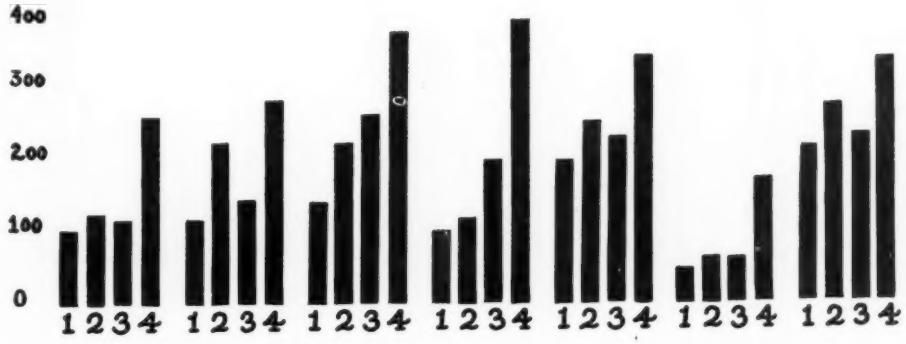

Chart 5

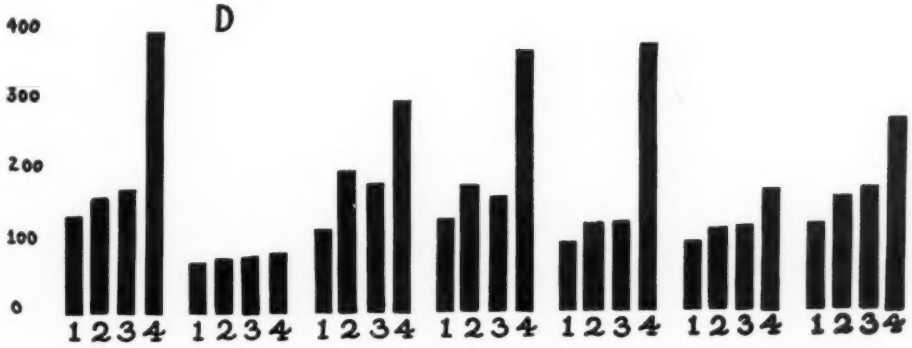

Chart 6

Chart 7

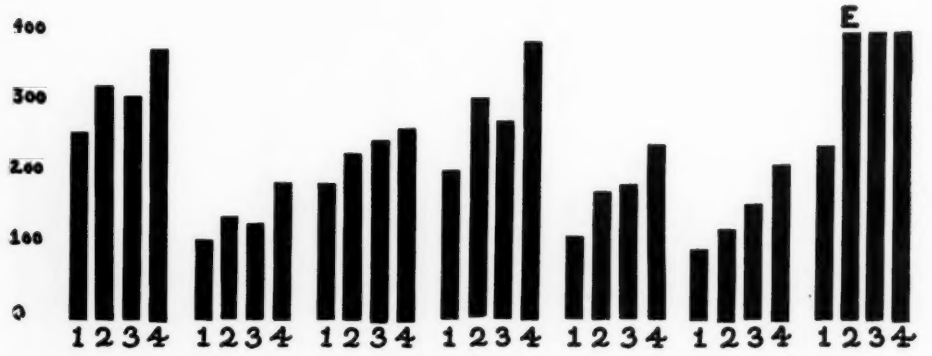

Chart 8

on the part of the patient. Coughing, holding the breath, grunting or even moving the head or body causes an increased intrathoracic pressure which dams back the blood in the internal jugular veins often as completely as compressing both jugulars, and thus vitiates the test.

(4) Normal difference on the two sides. They state that there is often a marked difference in the cerebrospinal fluid pressure when the two jugulars are compressed separately, but that a difference of over 50 mm. is unusual and over 100 mm. is exceptional; that, given a normal initial spinal fluid pressure, the rise on jugular compression of one side usually will be ample, seldom less than 50 mm., more probably over 100 mm. They noted, too, that the rise in pressure tends to be less when the jugular on the dependent side is compressed, and that occasionally variations in the anatomy of the tributaries to the internal jugular have an effect. In this respect the facial vein is most important. It is the blocking of the flow in the facial vein on jugular compression, with blocking of the ophthalmic venous system, which accounts for the slight rise in cerebrospinal pressure usually seen even when the sinus on the side being compressed is completely occluded by a thrombus. This factor, present in an unusual degree, must account for Kopetsky's observation.

The charts shown bear out these observations of the originators of the test.

CASE REPORTS

Case I.—R. A. W., a white boy six years old, was admitted June 26, 1927. A simple right mastoidectomy had been done two years before for chronic purulent otitis media. There was temporary improvement, but the right ear had been running again for nine months. For two days before admission he had complained of pain in and around the right ear. His temperature was 102°. His right ear was discharging profusely through a large perforation and there was redness and swelling in the region of the mastoid scar. The old wound was opened by the resident surgeon. No bony necrosis was noted. A mass of pus and granulations were scraped out of the antrum. The sinus and dura were not exposed. After a week his temperature rose and on July 6 it reached 105°. The leucocyte count was 12,000. There was no evidence of meningitis and no nystagmus. The spinal fluid was clear, its pressure within normal limits with 6 cells per cu. mm. The

Tobey-Ayer test was positive. Pressure over the left jugular vein made the fluid run out at the top of the manometer. Pressure on the right jugular caused no rise. At operation a perisinus abscess was found. The right internal jugular vein was cord-like. It was cut between ligatures. The sigmoid sinus contained a clot. I was able to get free bleeding behind but not below. Blood cultures taken on the fifth and sixth were later reported negative. A transfusion was given on the eighth. The course of the sepsis was not changed, however, and the patient died on July 19. Permission for an autopsy was refused.

In this case I should have excised as much as possible of the jugular vein, as is recommended in cases in which the vein is visibly affected.[5,6] I did not do so because the vein was not so much thrombosed as collapsed. I thought that the septic process was above the ligature. In this case the information given by the Tobey-Ayer test was perfectly definite.

Case II.—V. L. E., a white boy eleven years old, admitted July 12, 1927, had had trouble with his ears all his life. Four days before admission he had a right myringotomy.

On admission, his temperature was 103.5°, his leucocytes 16,000. The right ear was discharging copiously. The left ear drum was intact and retracted. The right mastoid was very tender. The anterior cervical lymph nodes were swollen and his neck was somewhat stiff. The Kernig and Brudzinski signs were negative. The spinal fluid pressure was high, 200 mm. The fluid was clear and had ten mononuclears per cu. mm. The Tobey-Ayer test was negative.

A right simple mastoidectomy was done at once. The cells were full of pus and granulations. The sinus plate was carefully outlined and it looked healthy. The sinus was not exposed. The temperature remained elevated without marked remissions and at the end of a week it was definitely septic, see-sawing from normal to 105°. At operation, on July 19, the internal jugular vein was patulous. It was divided between ligatures. The sigmoid sinus was also patent and bled freely above but sluggishly below. A parietal bulbar clot was evidently present. On July 20, the right orbit and the right side of the face were greatly swollen. A blood culture was reported positive for *Streptococcus hemolyticus*. The septicemia could not be controlled, and he died after a convulsion on July 29. Autopsy was refused.

Comment.—The Tobey-Ayer test was negative in this case and at operation no obliterating clot was found. The diagnosis rests on the septicemia with positive blood culture and no demonstrable lesion save the otitis media[7] and mastoiditis and[2] the markedly sluggish bleeding from the bulbar end of the sigmoid sinus.[8] The sinus and jugular operation was deferred too long. A negative Tobey-Ayer test does not exclude a mural thrombosis.

Case III.—Miss R. P., a white girl 16 years old, was admitted on the medical service February 4, 1929. This patient is an almost total diabetic who has spent a large part of her life in the University Hospital. Her complaint at the time of this admission was an upper respiratory infection with fever and headache of two weeks' duration. On the third day after admission both eardrums required incision. Fifteen days after admission the patient began to run a high temperature which reached 105° two days later. A bilateral simple mastoidectomy was done on February 23. Very little pus was found, but there was great vascular engorgement. The temperature was lower for two days, but on February 27 and 28 it reached 104°.

On February 28 the Tobey-Ayer test was done and it was found that compression of the left jugular vein caused a prompt rise of 60 mm. Accordingly, about 2 inches of the left internal jugular vein was excised. It was not thrombosed. No clot was found in the left sigmoid sinus. The mastoid emissary vein was about 3 mm. in diameter. It entered the sinus rather lower than usual and so near the bulbar end of the exposure that it was impossible to distinguish between bleeding from it and from the inferior petrosal. A blood culture taken two days before the second operation was later reported positive for *Streptococcus hemolyticus*. Improvement was prompt. The first day after the resection the temperature reached 102.2° and it was lower each successive day. A blood culture taken several days after the operation was negative. Three weeks later an abscess in the patient's right thigh was opened. It yielded, on culture, *Streptococcus hemolyticus*. The mastoid and neck wounds healed well. This patient has been in the hospital twice since this siege for her diabetes. Her ears seemed normal.

Case IV.—L. T., a colored woman 26 years old, was admitted to the University Hospital on March 16, 1929. Five days before admission much pus was evacuated by incision of a right peritonsillar abscess. Three days later she developed a severe pain in her lower left chest. She was admitted on the medical service complaining of pain in her left side. Her temperature was 104.5° and her white cell count 11,400. Her ears were not examined at that time, but she specifically denied in her history any earache, aural discharge, tinnitus, or deafness. Her right tonsil was enlarged and there was a healing incision above it. The anterior cervical lymph nodes were swollen and tender. The chest examination was negative. A blood culture was later reported positive for *Streptococcus hemolyticus*. X-ray examination showed a small area of apparent consolidation near the base of her left lung. There was no evidence of a subphrenic abscess. Her urine contained hyaline and granular casts for two days. For two days her temperature went above 104° with a leucocyte count of 20,000. On March 19 and 20 its maximum was about 102°. The next day she had a chill and a temperature of 104.2°. On March 22 she received 225 c. c. of blood. Her white cell count was 16,000. On March 23 her ears showed no abnormality except slight retraction of the drums and a slightly enlarged and tender post-auricular lymph node. There was no deep tenderness. The Tobey-Ayer test showed no rise of spinal fluid pressure when the right jugular vein was compressed. Compression of the left jugular caused a rise of more than 100 mm. That afternoon the neck was explored. No pus was found, but the right internal jugular vein was collapsed and apparently solid. It was resected between ligatures from a point about five cm. below the facial branch to 1 cm. above the facial. There was a clot at the end of the facial, so 1 cm. of the facial vein was excised. For three days there was apparent improvement, though a blood culture taken the day after the operation was

positive for *Streptococcus hemolyticus*. Her maximum temperature on March 26 was 99.8° with 10,000 leucocytes. The next day her temperature went to 102.2° with her leucocytes more than 13,000.

It seemed likely that the clot above the excised bit of vein was infected and was causing sepsis. At this time the right ear drum was negative, but there was deep tenderness over the emissary vein. Accordingly the mastoid was opened. The antrum and the aditus were normal. The zygomatic cells and all the anterior cells were also normal. Several cells adjacent to the sinus, however, contained pus. When the sinus had been opened a clot approximately 6 cm. long was removed. There was free hemorrhage from each end. This clot contained non-hemolytic streptococci. Blood culture taken after the operation was negative. Recovery was uneventful except for contraction of scar tissue in the neck which required physiotherapy.

Comment.—This case is interesting on account of the rarity of the pathological sequence. I have been able to find reference to only five similar cases in the American literature. Tobey,[4] in 1926, stated that he had never observed and could not find authentic instances of lateral sinus thrombosis in the absence of aural disease. He quoted Trendelenberg (1908) and Mosher[9] (1921) as having observed thrombosis of the internal jugular vein as a result of pharyngeal infection. Of course in this case that was the first step. Retrograde thrombosis of the sigmoid sinus was the second step.

In Long's[10] case, reported in 1912, there was sore throat, followed by sepsis with thrombosis of the left internal jugular vein which evidently extended up to the cavernous sinus. The vein was excised from the clavicle to the jugular foramen and the patient recovered after a long illness with no intra-cranial intervention. Goodman's[11] patient also recovered without having her cranium explored. Practically the whole internal jugular was excised. No autopsy was permitted in Mosher's[9] case. At operation, however, an abscess was found in the neck and the internal jugular vein could not even be identified with certainly. In Kernan's[12] case the autopsy showed thrombosis not only of the jugular vein and lateral sinus, but the superior and inferior petrosals and both cavernous sinuses and even the ophthalmic veins. There was also a brain abscess. Eagleton's[13] "Case F" had autopsy findings like that of Kernan's patient.

The important thing in these cases seems to be early recognition of the jugular thrombosis and also of the probability that the sigmoid sinus will be secondarily involved. In making the diagnosis in this case the Tobey-Ayer test was of pre-eminent importance.

Case V.—J. S., a white boy 2 years old, was admitted to the Martha Jefferson Hospital on Sept. 8, 1929, with a septic sore throat and left acute mastoiditis. Dr. Woodward did a simple mastoidectomy at once. The child had a moderate fever for ten days, when the temperature became typically septic in character. At this time there was a rather erysipeloid rash around the wound. Previously when the skin clips were taken out the wound gaped open and it was noted that there was no tendency to wound repair. No granulations were to be seen fifteen days after the operation. The Tobey-Ayer test, done on the fifteenth day, was absolutely inconclusive, whenever pressure was applied anywhere, whether over one jugular or both or the back of the neck he cried harder and the pressure went up to its maximum. The next day the rash faded, but the day after that the temperature reached 106.4° and Dr. Woodward ligated the left internal jugular vein and opened the sigmoid sinus. No clot was demonstrable and bleeding was very free from above and quite free below. The temperature reached 104° daily for twelve days after the ligation. Multiple abscesses in the scalp were opened and drained during this time. At last the temperature fell gradually and the child was sent home October 13 to have his wounds dressed by his family physician.

Comment.—This case shows the impossibility of obtaining satisfactory manometric readings when a child is crying.

Case VI.—E. L., 70 years old, a colored preacher, was admitted to the University Hospital on Nov. 13, 1929, complaining of severe pain behind his left ear, vertigo and left facial paralysis, all of two months' duration. He gave a history of having had a discharge from his left ear and from a sinus behind the ear for 60 years. The left ear drum was destroyed. It and a sinus behind the ear at the junction of the auricle and scalp one inch above the external auditory meatus were discharging a sero-mucous fluid. There was complete left facial paralysis. Irrigation of the right ear with water at 68° gave a quick nystagmus after 15 seconds. Irrigation of the left ear caused no nystagmus. The left ear was totally deaf. The cerebrospinal fluid was normal, its pressure not increased. Compression of the right jugular caused a quick rise of spinal fluid pressure of 115 mm. Compression of the left jugular caused a slow rise of 50 mm. At operation the roof of the middle ear and mastoid antrum were entirely destroyed and these two cavities, the latter of which communicated with the sinus behind the ear, were completely filled with granulations from the dura of the middle fossa. The sinus plate was hard, but on its removal it was found that there was thickening as from old granulations along the line of the sinus, and that the sinus seemed to be obliterated. It is too soon to say what the outcome in this case will be.

Comment.—In this instance the sinus had probably been obliterated for many years. The collateral circulation being well established, partly through the facial and ophthalmic veins, when the Tobey-Ayer test was done there was a marked slow rise of pressure, but not half as high when the left jugular was compressed as when pressure was made on the right.

CONCLUSIONS

(1) Within the limitations set forth by its authors in patients without an obliterating thrombus of the sigmoid sinus or internal jugular vein, the Tobey-Ayer test gives negative findings.

(2) In patients with obliterating thrombosis the test is often of supreme value.

(3) A mural thrombus may give negative findings. Hence, if the other evidence in favor of thrombo-phlebitis is strong, operation should not be too long deferred.

(4) The test is wholly inconclusive in crying children.

(5) When the occlusion of the sigmoid sinus is of long duration and the collateral circulation has become established there is a slow rise of cerebrospinal fluid pressure when the jugular on the affected side is compressed, but probably not as great a rise as occurs when the opposite jugular vein is compressed.

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DISCUSSION (*Abstract*)

Papers of Dr. Marks and Dr. Tunstall.

Dr. Murdock Equen, Atlanta, Ga.—Those of us who have written papers must realize the enormous amount of work entailed in preparing these charts, and we certainly should appreciate the contribution of Dr. Tunstall. The care of a patient having a condition in which sinus thrombosis is suspected is a most responsible one, and requires the assistance of the internist, neurologist, and bacteriologist. Each must work hand in hand with the other in deciding whether or not the case should be operated upon, or whether to assume an air of watchful waiting. Now these charts illustrate well the findings on compression of the internal jugular veins where a complete obstruction occurs. However, we must remember that a negative Tobey-Ayers test does not in itself rule out sinus thrombosis, as we may have a mural clot which permits the passage of a certain amount of blood. In making this test we must have complete cooperation of the patient, and it is frequently best, especially with boisterous children, to anesthetize them. In the presence of a mastoiditis accompanied by a bacteriemia, usually a streptococcus bacteriemia, in which the patient has a high white count, high fever, and positive blood culture, surgical interference should be instituted immediately. Blood transfusions are of distinct value. I have seen several cases with definite symptoms of sinus infection which rapidly cleared up following a transfusion. In cases that are operated upon there is always an accompanying loss of the body fluids which must be made up. The only way to replace this fluid is by the administration of saline and glucose, or by blood, whole or citrated. I prefer whole blood transfusions.

Dr. F. E. Hasty, Nashville, Tenn.—Dr. Marks' case reports recall to one's mind the variation in the severity of cases of otitis media that accompany different epidemics of influenza or colds. During an epidemic in which the ear complications are caused by a variant infection, it is well to open the ears as soon as there is any evidence of bulging. In my experience, it is sometimes necessary to open the ear drum more than once, but I think that one should be rather cautious about opening an ear drum repeatedly.

It is not advisable to do a mastoid operation early in any case, and surely not in those cases where there is a *Streptococcus hemolyticus* infection. My feeling is that if the patient cannot offer a certain amount of resistance to the invading organisms, the surgeon will only further jeopardize the patient's life by operating before Nature has built up a local or systemic resistance. I am aware of the fact that some surgeons advise mastoid surgery within a week or ten days after the middle ear is first involved. I seldom advise surgery earlier than twelve to fourteen days after the original infection.

The diagnosis of septicemia of otitic origin is a process of elimination from beginning to finish. When a well supported presumptive diagnosis of septicemia has been made, immediate surgery is indicated; but the surgeon should always remember that of all the cases presenting the one most common evidence of septicemia, namely, a marked rise of temperature, only a small percentage will eventually prove to be septicemia. Pneumonia is one of the most common conditions that will at first arouse the fear of septicemia, and indeed with the pneumonia there may be bacteria in the blood stream. It is my practice when competent internists even suspect pneumonia, to have an x-ray of the chest every twelve to twenty-four hours, made by the bedside unit, until the diagnosis is made. If competent laboratory service is available, the surgeon can frequently get valuable information by a study of the blood, but it should be remembered that with the best of laboratory

accommodations, in not more than 50 per cent of the cases can a positive culture be secured.

The surgery should be rapid, accurate and complete.

Lateral sinus thrombosis should not be treated by a surgical meddler. The vein should be dealt with first. The lateral sinus should be widely exposed, the plug should be placed at least one-half inch posterior to the apparently diseased area. Once the surgery is done, one should be very slow indeed to repeat any operative procedures. The secret of success lies in reserving and building up the patient's resistance to the invading organisms, and in such conditions every surgical undertaking taxes rather heavily the patient's power of endurance and resistance.

I prefer whole blood transfusions in rather small quantities, and these not to be used when the hemoglobin is above 60. I have seen hearts dilate when large quantities of fluid were introduced into the blood stream. It should be remembered that these patients are capable of taking adequate fluids, and that the transfusion is done to fortify the blood. Unnecessary examinations and laboratory tests of one kind or another use up the patient's resistance, and therefore decrease his chance of recovery.

In my experience patients under twelve years of age are poor risks. About 33.33 per cent recover. The younger the patient, the less the chance of recovery. Likewise the aged stand the infection very poorly. Young adults or middle aged people should show a recovery of about 70 per cent. One may have a small series of cases with a high percentage of recoveries. On the other hand, one may have a series with a low percentage of recoveries. Much depends upon the virulence of the organism and the resistance of the patient. Poor management may easily destroy the patient's chance of recovery.

Dr. J. W. Downey, Baltimore, Md.—I am exceedingly interested in Dr. Tunstall's work because I heard Tobey's original paper and since then I have followed the test very carefully. I have had a number of cases in connection with Dr. Charles Bagley, of Baltimore, who is as you know one of our outstanding brain surgeons. One of the nicest things in his work is his gentleness and kindness with patients, particularly with children. I believe that if any man can keep them quiet, he can. And yet we have often seen the test rendered uncertain by an unavoidable movement or outcry.

We must not let this test, scientifically and theoretically beautiful as it is, obscure our clinical observations in these infections.

Often the diagnosis of lateral sinus thrombosis must be made by a process of elimination, but I do not feel that we are justified in waiting too long nor depending too much on the test we are discussing, especially in unilateral cases of mastoiditis. Certainly these are suggestive local signs to guide us. The mastoid wound is usually unhealthy, the dural wall of the sinus covered with granulations or showing an ulcerated area and the neck over the jugular is apt to be tender. In double mastoiditis with symptoms of thrombosis, both sinuses should be fully exposed and a careful search made for even the smallest spot of ulceration of the dural wall, which may guide us as to the side which should be operated upon. May I cite one case which brings out this point and also shows that we cannot depend entirely on the Tobey-Ayers test? A boy of twelve had double mastoiditis and symptoms of lateral sinus thrombosis. The right sinus was covered with dirty granulation and the dural wall seemed thickened and inflamed. The left sinus appeared normal. The Tobey-Ayers strongly indicated involvement of the left side and we operated upon that side. We could not demonstrate even a mural clot. There was no abeyance in symptoms. Several days after, the right sinus ruptured spontaneously during a dressing. We got out a large infected clot and packed off as quickly as possible when hemorrhage became profuse. Following this, the youngster began to improve and, though he developed a metastatic abscess in the knee, he got well.

I most emphatically disagree with the opinion that we must wait for a positive blood culture before we operate. If we do most of our cases will die. When we can make a comparatively early diagnosis the prognosis is by no means unfavorable.

Dr. Oscar Wilkinson, Washington, D. C.—In my experience there have been just three things which have been particularly helpful in infection of the lateral meninges. In the first place, as Dr. Hasty said, thorough, clean and quick surgery should be done when you do surgery. That is the first essential. The second is that a small, supporting transfusion should be given, whether you use whole blood or citrated. I think probably whole blood is better. But many very sick patients have been harmed by having too large transfusions at one time, interfering with the support of the heart. In other words, instead of supporting the patient you have added an additional load. The third thing that has been of service to me is spinal drainage. Where you have a complicating infection in the brain from thrombosis of the lateral sinus, spinal drainage has been of marked benefit in one or two cases and it has been an undoubted aid in the recovery of those cases.

Dr. Marks (closing).—I did not wish to convey the idea that every case of streptococcic otitis media with severe systemic manifestations should have a mastoid operation; but that when mastoid involvement is demonstrable by x-ray and evidenced by increasing toxemia, an operation should be done.

In this type of case we should be constantly alive to the possibility that meningitis may arise, and we should be early prepared to use to the best advantage any treatment to lower its high mortality.

We must not depend upon the blood culture. The first case reported had never had a positive culture, but unquestionably had a blood stream infection. Careful study of these cases is important. Every case in which there was a suspicion of pneumonia was x-rayed. Those cases in which pneumonia was not suspected were not x-rayed. But the case of hemorrhagic otitis media with general sepsis of the chest was x-rayed twice before we operated, and we decided there was nothing there. The pneumonia probably developed later.

The laboratory is always a valuable assistant, but we cannot depend too much on it. One must see the x-ray pictures for careful study and conclusions.

Rapid surgery is desirable. About forty-five minutes is as little time as I find necessary to expose the sinus, tie off the vein, and close.

So many procedures have been advocated that one is often seriously puzzled as to the best thing to do, but must endeavor to choose the procedure that will help the greatest number of these patients. I believe we were justified, particularly in this second case, because we did find a mural clot on one side and on the other side a

phlebitis. I do not know whether the other patients had a phlebitis or not. These are bad cases, and the case that gets well is the one that is able to withstand the streptococcus, as Dr. Hasty has said.

Dr. Tunstall (closing).—In regard to the charts (pages 640, 641 and 642), this test (A) would be inconclusive. The patient has as high pressure on compression of the left jugular as on compression of both. There must have been some error in technique in that case, because this (B) is the same patient done a few days later and the findings are different. I do not know just what the error is. These tests were done by another department, and in their routine procedure they just noted for me the results of these tests. They were done partly by students and partly by the internes, but I am sure there was an error there. That is the sort of thing Dr. Downey spoke of, and how they can fool one.

C is a case of spinal cord tumor with complete blockage of the subarachnoids. Cases like this (D) are very few, where there is low intracranial pressure. One does not get much increase of tension where the spinal fluid pressure is originally low. In this case (E) the fluid went to the top of the manometer and we do not know how much higher it would have gone. It would have to be done over with a longer tube.

I agree with Dr. Hasty and with several of the other physicians who have spoken, that we cannot afford to wait for a positive blood culture. It may take too long. The sooner we make the diagnosis the better. In the series which I presented, we have only two or three in which the blood culture report was in before the diagnosis was made.

I have had no experience with spinal drainage and do not quite understand how it would help.

OXYGEN THERAPY IN EPILEPSY*

By PAT MURPHEY, M.D.,
Little Rock, Ark.

I shall not attempt to discuss the symptoms of either idiopathic epilepsy or the epilepsy that follows organic brain injuries, such as that found in infantile cerebral palsy. Rather, it has been my hope that I might find some means of relieving these conditions. I have conducted some experiments in oxygen therapy, using as my subjects several patients in the State Hospital for Nervous Diseases at Little Rock, with the full consent of both the hospital authorities and the patients involved. These patients are all men who have been confined in the hospital for some years and represent both types of epilepsy.

On the ward as the patient had the epileptic attack, I immediately applied the oxygen mask to his nose and mouth so that he breathed the gas in as soon as he took a breath. This was tried on eight patients, five idiopathic and three organic, and in each case I found that relaxation began as soon as a breath was taken and at the end of two or three inhalations the man was completely relaxed and the cyanosis gone. Also the drowsy period which usually follows such attacks was materially shortened.

In some instances, however, several seconds elapsed between the time that the mask was applied and the time the man took a breath, due to his convulsive state. In an effort to offset this loss of time, I decided to give the oxygen intravenously. Accordingly, I have injected pure oxygen directly into the veins of twelve patients, ten being idiopathic and two organic cases. It has been repeated in several cases on different days as I happened to see them in other attacks. Some of these men had already been given the oxygen by inhalation.

The gas was given 30 c. c. at a time and the men received a total dosage of from 90 to 465 c. c. No bad effects at all were seen in any of the cases. One man was made drunk and reeled as he walked for a few minutes. Six other men developed coughs after taking from 120 to 445 c. c., but this subsided shortly after the oxygen was stopped. This seemed to prove that the oxygen went through the heart into the lungs and the irritation there set up the cough. To substantiate this, x-ray pictures were made of two men after oxygen had been injected into their veins. The oxygen could be traced from the point of introduction to the shoulder and there was lost. It appeared on the film as a narrow radiolucent streak not more than 2 mm. in width at any point.

CASES

Case 19597.—H. E. B. was admitted to the hospital in 1925, at the age of eighteen, for a mental condition following fits. No heart nor lung disease nor deformities were found. Blood examination and urinalysis showed no evidence of disease. He had the usual diseases of childhood and had had epileptic spells for at least five years. Dating from the time of his admission, he had had them every few days. His hospital diagnosis was mental deficiency without a psychosis, epilepsy, *grand mal*.

I saw this man on September 15, 1929, in a convulsion. He was sitting on the bench and without warning became unconscious and at once began to jerk and show contractions in his arms and legs that flexed him. When we got him on the floor, his whole body was in a general tonic convulsion. He was blue, cyanotic, and not breathing. His chest was fixed. The oxygen mask was applied to his nose and mouth. In a few seconds he took a breath, inhaling the oxygen, and immediately relaxed. Then he began to breathe regu-

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larly and the oxygen was continued for about one minute when the cyanosis disappeared. He had an incontinence of the bladder before he was relaxed. He got up off of the floor and was able to sit on the bench in six minutes from the time his convulsion started. The attendant stated that this was the shortest attack that this man had ever had and that the ensuing drowsy period was cut short, as he usually lay on the floor a half hour.

Again on September 21, 1929, without warning, he became unconscious. The contractions of the arms and legs were not so hard as they were on the fifteenth. The first inhalation of oxygen began to relax him and two or three breaths brought him out of his convulsion with the cyanosis gone. He was able to get up on the bench four minutes after the onset of his attack.

Case 18707.—Roy W. was admitted to the hospital in 1924, at the age of thirty-seven. He had epileptic convulsions all of his life. Physical examination revealed no heart nor lung disease; his blood pressure was 115/70; abdomen negative; he showed no atrophy nor anesthesia and no pathological reflexes were elicited. There was no paralysis nor paresis of cranial nerves. He gave a history of having had his first convulsion about four years before his admission to the hospital. During his attacks he fell to the ground, frothed at the mouth and bit his tongue. Often he had four or five a week, and he often slept for an hour after the attack. The convulsions occurred without aura. The blood Wassermann was negative and the urinalysis and blood picture revealed no disease of the kidneys or blood stream. The diagnosis was mental deficiency without psychosis, epilepsy, *grand mal.*

On September 15, 1929, I saw this man have a light convulsion. He lost consciousness and had tonic and clonic contractures of the arms and legs. By the time I got him on the floor and the oxygen mask over his nose and mouth, he had begun to relax, but was still blue and cyanotic. He took a breath as soon as the mask was in place and immediately relaxed. The oxygen was continued for about one minute after he was relaxed, when the cyanosis completely disappeared. He lay on the floor for five minutes and then got up and sat on the bench. The attendant states that his drowsy period was shortened.

Case 14137.—O. McF. was admitted in 1920, at the age of eighteen. He had manifested symptoms of mental derangement for twelve or fifteen years, with epileptic fits. He had a mitral lesion. Breathing was free and easy and expansion equal. There was no lung disease. His blood pressure was 122/92. Neurological examination revealed unequal pupils, irregular in outline; spastic paralysis of the right arm; partial paralysis of the right leg; no sensory disturbances and no ankle clonus. The face was asymmetrical. He had a right hemiplegia. He had been paralyzed all of his life and had had convulsions ever since he could remember. Just before he had an attack he said that he felt as if something were running up and down his left leg. Sometimes the attack was light and he did not fall. At other times he fell, frothed at the mouth, and bit his tongue. He had a fainting spell two or three times a week. He was mentally defective. The diagnosis was infantile cerebral palsy with epilepsy, mental deficiency without a psychosis.

I saw this man on September 15, 1929. He was sitting on the bench with the other patients, when he suddenly became unconscious and had a generalized convulsion. By the time we got him on the floor, he was holding his breath, was blue and cyanotic. He was in a typical tonic convulsion. The oxygen mask was put in place, but it was a few seconds before he took a breath. The first time that he took a breath he began to relax and by the time he took a third inhalation he was completely relaxed and the cyanosis was gone. He was drowsy and in a little stupor for ten minutes and then was able to get up. His ward physician said that his drowsy period was much shorter than usual.

Case 10985.—B. G. was admitted to the institution in 1917, at the age of twenty-eight. He had manifested symptoms of mental derangement for six years, and had had convulsions. His heart and lungs were negative. The abdomen was negative. There were no deformities nor contractures. There were numerous scars over his head, evidently from injuries during convulsions. Neurological examination revealed no anesthesia nor atrophy; no pathological reflexes were elicited and no paralysis nor paresis of any cranial nerve. He was deaf and dumb, and unable to read or write. The history did not show just how long he had had convulsions, but he had been observed in numerous attacks since his admission to the hospital and every time he fell, he completely lost consciousness, bit his tongue, and slept or remained drowsy for a half or whole hour after the attack. The diagnosis was developed mental defect with epilepsy.

On September 21, 1929, he fell to the floor in a hard convulsion. The oxygen mask was placed over his nose and mouth, but it was a few seconds before he took a breath. At the first inhalation, he began to relax and at the end of the fourth breath he was completely relaxed and the cyanosis had disappeared. The oxygen was continued for a few minutes more. This man was able to get back on the bench twelve minutes from the beginning of his convulsion.

Case 6713.—J. W. was admitted to the hospital in 1913, at the age of thirty-five years. He was said to have been injured and to have had an operation on the left side of the head. He had been paralyzed since that time. He had a luetic infection in 1897. His heart and lungs were negative. There was a right hemiplegia with a transitory motor asphasia; there were exaggerated reflexes on the right and partial paralysis of the muscles of the face on the right. There was a depression on the left side of the skull about the size of a silver dollar. This man stated that he had had convulsions since 1913, the year of his injury and operation. His blood Wassermann was four plus. Urinalysis was negative. He had convulsions without warning, fell to the ground and bit his tongue. The diagnosis was epilepsy, *grand mal,* traumatic, and luetic infection.

On September 21, 1929, without warning, this man lost consciousness and had a generalized convulsion. The oxygen mask was applied over his nose and mouth, but it was a few seconds before he took a breath. At the first inhalation he began to relax and by the third breath, he was completely relaxed and the cyanosis gone. He then responded to questions, three minutes after the onset of his convulsion. It was the opinion of the physician and attendant that this patient's convulsion was shortened by the administration of oxygen.

Case 19927.—R. B. was admitted in 1925, at the age of twenty-three years. He had manifested signs of mental derangement for fourteen years, and was absent minded. He began to have convulsions when he was a

small boy. He had no heart nor lung disease. All reflexes were present and active. There was no anesthesia nor atrophy, and no paralysis nor paresis of the cranial nerves. The blood Wassermann was negative. He had convulsions, both during the day and night, at irregular intervals, in which he fell to the ground and bit his tongue. The diagnosis was epileptic psychosis.

I saw this man in a convulsion on September 15, 1929. Without warning he became unconscious and had a generalized convulsion. It was about thirty seconds before we could get him to breathe after the oxygen mask was applied, but at the first inhalation he began to relax. After the second breath he was relaxed and the cyanosis gone. The oxygen was given him until he had breathed it six or seven times, when he began to pull at the mask. Within ten minutes he was able to get up.

Case 22638.—Robt. W. was admitted in 1927, at the age of forty-four. He had manifested symptoms of mental derangement all of his life, and had had convulsions. He was well nourished. He had no heart nor lung disease, but was paralyzed on the right side. He had right hemiplegia. There was no anesthesia. There was atrophy of the right arm and hand, and exaggerated reflexes on the right. There was no paralysis nor paresis of the cranial nerves. The blood pressure was 128/84. He gave a history of having had convulsions nearly all of his life, in which he fell and became unconscious. His right side jerked more than the left. He had residuals of infantile cerebral paralysis involving the right side. The diagnosis was infantile cerebral paralysis and epilepsy.

On September 21, 1929, this man became unconscious without warning and had a generalized convulsion. He did not get a deep breath after the oxygen mask was put in place for a few seconds. However, at the end of the fourth inhalation, he was completely relaxed and the cyanosis cleared. He was able to get up four minutes after the beginning of his convulsion.

Case 240053.—B. B. was admitted in 1929, at the age of seventeen years. He had manifested signs of mental derangement for eleven years. He began having epileptic seizures at the age of six. His heart and lungs were negative. His blood pressure was 120/75. His abdomen was negative. All reflexes were present and active. No pathological reflexes were elicited. There was no atrophy nor anesthesia, and no paralysis nor paresis of the cranial nerves. He had convulsions at irregular intervals without warning. His blood Wassermann was negative. The diagnosis was mental deficiency, epilepsy, *grand mal.*

On September 22, 1929, without warning, he lost consciousness and hit the floor just as if he had been shot or knocked down. The contractions in his arms and legs were tonic. The oxygen mask was applied and he took a breath just as soon as it was over his nose and mouth. His convulsions increased in severity until the second breath was taken. Then he began to relax and was entirely relaxed at the end of the fifth inhalation. He was in a stupor or drowsy condition for thirty minutes before he could get up. In an hour and a half this man was in a bathroom and had another convulsion. He fell on the floor. His convulsion had begun to subside by the time I got him where I could administer oxygen, but he was still blue and cyanotic. The first inhalation caused his contractures to disappear and the cyanosis to clear. This time he was unconscious eight minutes before he was able to get up and sit on the bench.

When oxygen is inhaled it must pass through the alveolar sacs to get into the blood. To eliminate this delay, I decided to give the oxygen intravenously. Some of the following cases are patients who have already been given oxygen by inhalation and in those instances I shall not repeat the history. They can be identified by their initials and case numbers.

Case 19597.—On September 29, 1929, I saw H. E. B. become unconscious and go into a generalized convulsion which did not seem to be quite so severe as the previous one. By the time I got him on the floor he was in a tonic convulsion and unconscious. Thirty c. c. of oxygen were given him in the veins with no change noted. Another 30 c. c. were given him, when he became relaxed. Another 30 c. c. were injected and the cyanosis disappeared. Still another 30 c. c. were given, making a total of 120 c. c. In about one minute after this was completed the man got up laughing and talking and walked over and sat on the bench. We could readily see this man's color change, and the hands and fingers become pink. His hands are usually cyanotic and cold. His pulse was 80 when he got up. About eight seconds elapsed between each administration of 30 c. c. of oxygen.

Case 19927.—R. B., without warning, on October 5, 1929, became unconscious and had a generalized convulsion. It was about thirty seconds after the onset of the attack before I could get the needle into his arm. Then 30 c. c. of oxygen were injected. The cyanosis began to clear and he was relaxed. At the end of the second injection of 30 c. c. of oxygen, his pulse was 115 a minute. At the end of the third 30 c. c., he was able to notice us and responded slightly to shaking and questions. The pulse began to slow and was now 90, after he had had 90 c. c. of oxygen. He was drowsy and in a little stupor for ten minutes, then got up and sat on the bench.

Case 22638.—Robt. W., without warning, became unconscious and had a typical epileptic convulsion on September 5, 1929. Thirty c. c. of oxygen were given intravenously. The cyanosis began to clear and relaxation was noticed. After another 30 c. c., he was completely relaxed and the cyanosis was gone. Another 30 c. c. were given. His pulse was 90. Yet another 30 c. c. were injected, making a total of 120 c. c. He began to resist and the administration was stopped. He got up in four minutes.

Case 17724.—J. R. K., admitted in 1923, was thirty-seven years old. He had manifested signs of mental derangement for twenty-five years. His heart and lungs were negative. There was no paralysis nor paresis of any cranial nerve. All reflexes are equal and active. There was no anesthesia nor atrophy. The blood Wassermann was negative and he had no kidney disease. The diagnosis was epileptic psychosis with deterioration.

On October 6, 1929, this man lost consciousness and fell backwards. He was holding his breath, blue and cyanotic and in tonic convulsions. Thirty c. c. of oxygen were injected and no change was noted. Thirty c. c. more were given, whereupon the cyanosis began to clear and relaxation was noticed. Another 30 c. c. were given and the contractions in his arms and legs disappeared. He was still a little blue and cyanotic. Another 30 c. c. were injected. He began to cough. About 5 c. c. more of oxygen were given and as the cough

increased in severity, the gas was stopped. At this time the pulse was 90 and respiration regular. He got up off of the floor in one and a half minutes after we stopped the oxygen. His cough lasted eight minutes and then cleared. His pulse was 87 five minutes after the convulsion ended. I believe the blood became saturated with the oxygen and this caused the cough; in other words, the oxygen went through the heart into the lungs and there set up an irritation which caused the cough.

Case 14137.—O. McF. had a convulsion with no aura on October 12, 1929. He was blue, cyanotic, frothing at the mouth and making the gurgling noises frequently heard in these patients. Thirty c. c. of oxygen were given in his veins and he relaxed immediately. Another 30 c. c. were injected, when the cyanosis almost disappeared. Still another 30 c. c. were given. This man was able to get back on the bench in two minutes after the administration of the first oxygen, having had a total of 90 c. c. In an hour he had another convulsion. He was blue, cyanotic and frothing at the mouth. No oxygen was given, but he was watched to see if this convulsion would be as severe as the earlier one in which oxygen was given intravenously. This attack lasted one and a half minutes. He gradually cleared mentally, but not so rapidly as he did when he had oxygen. He was drowsy and in a stupor and lay on the floor for fifteen minutes before he was able to get up, whereas he was up in two minutes when he had oxygen.

On October 26, 1929, I again saw this man in a convulsion. He was relaxed when 20 c. c. of oxygen had gone into his veins. Oxygen was continued until he had 210 c. c., when he began to cough. Then the administration was stopped. Again I believe the blood became saturated with oxygen, some of it got into the lungs and this set up the cough. His pulse and heart action were not affected. The pulse was 84 and slowed to 78 in thirty minutes after the end of the convulsion. I think that the convulsion alone would have a tendency to quicken the heart action if oxygen were not given. His blood pressure was 126/80 in five minutes after the convulsion was over. In an hour it was 130/90. In another hour it was 130/90 and in still another hour, three hours after the convulsion, it was 130/90. The attendant reports that this man had a light convulsion in about three and a half hours after the one just reported.

Case 18707.—I saw Roy W. again on January 12, 1929, in a generalized convulsion, frothing at the mouth. Thirty c. c. of oxygen were given intravenously. The gurgling in his throat stopped and he began to relax and after 60 c. c. the cyanosis had disappeared. Another 30 c. c. were given and still another 30 c. c. He had now had 120 c. c. and asked us to let him get up. When he arose he could scarcely walk, but reeled from side to side like a drunken man. He began to scratch his head, saying that it itched. This itching stopped in ten minutes, but it was twenty minutes before he regained his steadiness of gait.

Case 6967.—W. M. was admitted in 1913, at the age of seventeen years. He manifested symptoms of insanity from the age of eight. He had some kind of fever at this age, to which the convulsions were attributed. His heart and lungs were negative. His pulse rate was 84, and systolic blood pressure 70. He had no paralysis nor paresis of the cranial nerves. All the reflexes were equal and active. There was no anesthesia nor atrophy. The diagnosis was developed mental defect, imbecility plus epilepsy.

On December 12, 1929, this man was brought in on a litter in a convulsion, blue and cyanotic. Thirty c. c. of oxygen were given intravenously and he began to relax. Another 30 c. c. were injected, then he became entirely relaxed and asked for a glass of water. Another 30 c. c. were injected. Then he got up and walked into the bathroom. In about thirty minutes he had another convulsion. He was frothing at the mouth. This time he was in another part of the ward when the attack came on. When he was brought to the oxygen machine and I put the needle in his arm, I noticed that he turned his head towards me. It struck me that, perhaps, we were dealing with a hysterical attack this time. I watched him for a few minutes and came to the conclusion that this was a hysterical attack. It was decided to give him oxygen any way to see what effect it would have on him. Thirty c. c. were injected with no change noted in his color or contractions. Thirty c. c. more were given and still another 30 c. c. Then he began to cough and resist. Oxygen was continued, 30 c. c. at a time, until he had had 360 c. c. He then began to cough and resist to such an extent that we had to discontinue. His pulse was 90 and respiration 20. Blood pressure was 100/62. He was put in a room by himself and every few minutes he had an attack, but we paid no attention to him and gradually the attacks stopped. He was then allowed in a room with several patients and he began again to have convulsions every few minutes. These were typically hysterical. The ward physician saw this man have his first convulsion today and is of the opinion that it was typically epileptic, but those following were hysterical. The oxygen relaxed him in his first attack, but had no effect on him in the later ones.

Case 24336.—I. H. was admitted in 1929, at the age of twenty-two years. He had manifested signs of mental derangement for three years, and had had convulsions for ten years. His heart and lungs were negative. His blood pressure was 130/80. The abdomen was negative. No pathological reflexes were elicited. There was no anesthesia nor atrophy, and no paralysis nor paresis to the cranial nerves. There were no blood nor kidney diseases. The diagnosis was mental deficiency with epilepsy.

I saw this patient on December 27, 1929, have a generalized convulsion. He was somewhat relaxed when he got the oxygen into his veins but was blue, cyanotic and unconscious. He was given oxygen, 30 c. c. at a time, until he had taken 270 c. c. Then he began to cough enough to interfere with the administration and it was stopped. He began to notice us in three minutes after the injection of the first oxygen. Blood pressure was 102/79. His ward physician and attendant said that this man usually had a series of convulsions when he had any, but this time he was up and on the ward again in eight minutes from the beginning of his attack and had no more during the afternoon. It may be that the administration of the oxygen at this time is what kept him from having his usual series of convulsions.

This man is one of two of whom x-ray pictures were made in an effort to trace the oxygen through the veins. He was cyanotic. One hundred and fifty c. c. of oxygen were injected into his veins. The fol-

lowing is the laboratory report on him, made before and after the injection of the oxygen:

	Red Blood Cells	White Blood Cells	Differential	CO_2	Hemoglobin
Before	4,675,000	5,750	75-24-1	78	80
After	4,750,000	5,680	76-23-1	86	98

Case 20361.—L. M. was admitted in 1926, at the age of eleven years. He manifested signs of mental trouble in 1921. He had a partial paralysis of the right arm and leg. He had had convulsions for several years. His heart and lungs were negative. His blood pressure was 95/55, and pulse 96. His pupils were irregular in outline. He had a right hemiplegia, unsteady gait, speech defect, coarse jerking movements of the right arm and ankle clonus on the right. He had no kidney nor blood disease. The diagnosis was infantile cerebral palsy with epilepsy.

This boy began to have convulsions during the night of October 29, 1929, and had them throughout the night from ten minutes to a half an hour apart. On the morning of the twenty-seventh he was still having them every thirty minutes. At one o'clock he began to have them from five to ten minutes apart. He was brought on the ward at about two o'clock and I saw him have a typical epileptic convulsion. He was partially relaxed when I put my needle into his arm. When I had given him 30 c. c. of oxygen, he began to have a convulsion but it lasted only a few seconds and then he relaxed again. Another 30 c. c. of oxygen were given and another 30 c. c. At the end of the fourth 30 c. c. he had another convulsion, but it was lighter even than the one just preceding and was mostly in the muscles of the face. The spasms of the face stopped when 240 c. c. of oxygen had been injected. I continued to give him oxygen, 30 c. c. at a time, until I had given 445 c. c. Then he began to turn blue around the eyes and mouth and I stopped. His pulse was 110 at this time. The first oxygen was given at three o'clock. At three-thirty he was clearing mentally and told us his name. At five o'clock he was able to sit up, drink a glass of water and ask for food. His blood pressure was then 98/65. He had no more convulsions on this day or during the night. His ward physician reports that he had seven convulsions on the morning of the next day. I believe that the administration of oxygen stopped this boy's convulsions. It took more to stop his attacks and clear him mentally than with any other patient who has had it.

Case 23448.—H. R. was admitted in 1928, at the age of forty-eight years. He had manifested signs of mental trouble for five years, and had had convulsions of the epileptic type for twenty-two years. He had no heart nor lung diseases. His blood pressure was 130/90. His abdomen was negative. The kidneys and blood stream were negative. There was no anesthesia nor atrophy; no paralysis nor paresis of cranial nerves. Neurological examination was essentially negative. The diagnosis was mental deficiency with epilepsy.

I saw this man on October 19, 1929. He became confused and drowsy and began to wander about the hall. Thirty c. c. of oxygen were given him intravenously about six or seven seconds apart until he received 150 c. c. Then his cyanosis disappeared and he began to cough. Oxygen was continued until he had had 240 c. c., when his cough began to interfere with the injections and it was stopped. He quit coughing in two minutes after the oxygen was stopped and was clear mentally in five minutes.

Case 24239.—J. M. H. was admitted in 1929, aged thirty-five years. He had manifested symptoms of mental trouble since the age of seven, and had had epilepsy. His heart and lungs were negative. His blood pressure was 124/80. His abdomen was negative. No pathological reflexes were elicited; there was no paralysis nor paresis of the cranial nerves, no blood nor kidney disease. The diagnosis was epilepsy, *grand* and *petit mal*, with deterioration.

On October 19, 1929, this patient had a convulsion. The contractions had almost stopped in his extremities, but his eyes were set and he was chewing his tongue. Thirty c. c. of oxygen were injected and he relaxed. Oxygen was continued in 30 c. c. doses until he had had a total of 445 c. c. Then he began to cough considerably, and oxygen was stopped. His cough disappeared in about four minutes and he got up off the floor and sat on the bench five minutes from the beginning of the administration of oxygen. According to the attendant this man was wont to lie on the floor from a half to a whole hour after his attacks.

Case 19712.—H. A. was admitted in 1925, forty-five years old. He had shown mental symptoms during the previous eighteen months. He gave a history of having had epilepsy since he was about eighteen years old. His heart and lungs were negative. His blood pressure was 110/70. His abdomen was negative. There was no paralysis nor paresis of the cranial nerves; no anesthesia nor atrophy. Neurological examination was essentially negative. The diagnosis was epileptic psychosis with deterioration.

On October 20, 1929, this man became unconscious and had a general epileptic convulsion. He began to relax when 30 c. c. of oxygen had been injected into his veins and was completely relaxed after he had had 60 c. c. He began to notice and regain consciousness when 120 c. c. had been given. He began to cough after 150 c. c., but 200 c. c. in all were given. This patient got up and was able to walk immediately after the needle was removed from his arm.

H. M. was the second patient who received oxygen for the purpose of x-raying. There is no cyanosis in this case. He was given 120 c. c. of oxygen intravenously. His laboratory report was as follows:

	Red Blood Cells	White Blood Cells	Differential	CO_2	Hemoglobin
Before	4,250,000	6,000	72–28	57.6	75
After	4,350,000	3,000	78–20–2	53.6	85

SUMMARY AND CONCLUSIONS

In epilepsy there is an oxygen and carbonic acid (fixed acids) imbalance in the cells. There is a gradual oxygen decrease, or lowering of oxygen pressure, until the fixed acids depress the cells beyond the point of functioning and at this stage consciousness is lost. The reflex and irritation to the motor areas cause the contractions of the extremities and the trunk.

In the beginning the irritation of the sensory nuclei of the cranial nerves and sensory areas of the cortex causes the sensory aura, to-wit: irrita-

tion to the seeing centres causes the flashes of light which the patient frequently sees just before loss of consciousness. The stimulation of the sensory nuclei (visceralis and sensibilis) of the tenth cranial nerve causes the sensory aura in the abdomen, heart, esophagus and throat: the sensation of a ball rising from the pit of the stomach to the throat and then loss of consciousness. Stimulation of the motor nucleus (nucleus ambiguus) of the tenth cranial nerve causes the cry-out in the patient just before loss of consciousness, when the motor impulse goes over the recurrent laryngeal nerve to the larynx.

I do not believe that it is the lesion itself in organic brain lesions that is responsible for the convulsions, but the imbalance between the oxygen and the fixed acids. The cells at the place of injury are more susceptible to the metabolic disturbance. It is the fixed acids in the cells that fail to be oxidized that cause the convulsion.

By the fixed acids, I mean the carbonic and sarcolactic acids. These acids normally oxidize into carbon dioxide and water in the presence of plenty of oxygen. Lactic acid penetrates the respiratory centres more than any other acid. Therefore it lowers the pH of the cells.

Therefore, oxygen given by inhalation or intravenously relaxes the patient in an epileptic convulsion, in organic brain lesions, and also in the idiopathic type by oxidizing the fixed acids in the cells. The drowsy period that usually follows such an attack is greatly shortened. No untoward effects were obtained from the giving of oxygen intravenously in amounts from 30 to 465 c. c. Pure oxygen was given by letting it flow from the oxygen tank into a syringe of 30 c. c. capacity. A three-way stopcock was used and this necessitated a lapse of six or seven seconds between each injection, while the syringe was refilling. The rapidity of the flow into the veins and the amount of pressure was controlled by the hand, just as in giving any intravenous medication. The blood usually took up the oxygen at the point of injection if it was given slowly. If given rapidly it could be felt for a distance of two inches or more from the point of introduction. I was able with x-ray pictures to trace the oxygen in the superficial vein in the arm from the point of injection to the shoulder and there it lost its identity. The cough obtained by giving oxygen intravenously, I believe, was caused by irritation in the lungs. Hemoglobin increased from ten to twenty points after the administration of oxygen.

I am convinced that the administration of oxygen not only hastens relaxation, stops the convulsion, causes cyanosis to disappear earlier and shortens the ensuing drowsy period, but that, in idiopathic cases, the attacks are fewer in number after the patients have had oxygen, whereas those of organic type remain much the same in number. I base this belief on records kept by attendants in the hospital of the number of convulsions among these patients mentioned above before and after the administration of oxygen.

DISCUSSION *(Abstract)*

Dr. W. E. Gardner, Louisville, Ky.—There is no doubt that in the fairly large series of cases which Dr. Murphey has reported, he has been able materially to shorten the stage of clonic convulsions, and the prolonged stage of stupor which follows the average epileptic seizure. If this alone can be done it will have a tendency to prevent secondary degenerative changes in the brain substance as the result of punctate hemorrhages which have been noted at the autopsy table and which have been thought to be a by-product of the convulsive manifestation rather than the cause of it.

I am not fully convinced that the frequency of the convulsions has been markedly reduced in any given number of cases extending over a prolonged period of time, and am unable at this time to see very much permanent benefit to be obtained from this method unless it might interrupt the habit tendency of the convulsion to repeat itself at more or less regular intervals.

Dr. Murphey's theory as to the underlying abnormality of the acid-base equilibrium in epileptics is not quite clear to me, but would seem to indicate that he is endeavoring to correct a condition of so-called acidosis of the brain cells which we have for the past several years been endeavoring to produce by the use of the ketogenic diet, especially in children, with whom such experiments seem to be most successful.

Much attention has been paid recently to the question of faulty water metabolism as an underlying factor in epilepsy, and there is a possibility that the administration of oxygen during the epileptic seizure may have some influence in correcting this fault, at least temporarily. It would seem, however, to counteract any tendency towards the production of an acidosis and would be more likely to produce a condition of relative alkalosis just as hyperventilation is thought to do in experimental epilepsy and some cases of narcolepsy which have been produced by this method.

In the *Archives of Neurology and Psychiatry* of July, 1928, W. G. Lennox reports his experiments in measuring the plasma bicarbonate in 100 patients with epilepsy, and in the large majority of these the acid-base equilibrium was normal. When abnormal, however, it was in the direction of increased alkalinity, and this is in keeping with the experiences of other workers along this line.

I shall await with great interest a report of Dr. Murphey's further experiments.

Dr. William Nelson, St. Louis, Mo.—It is said that one person in 25 of the general population has epilepsy. It would seem that anything developed in the direction

of lessening the frenzy and the tears of epileptic seizures would be welcomed by the medical profession as well as others, and whether or not such a procedure would have an instant influence in reducing such attacks, the mere question of lessening these psychomotor disturbances would be of considerable value.

We are all conversant with the effect of oxygen on the muscles. Occasionally while we are making an examination of the lungs a patient will become dizzy and faint for a short period. The influence of oxygen in quieting the motor spasm is something we have known for quite a long time.

Certain mathematical implications in the relaxation of the muscles hardly fit in with Dr. Murphey's scheme for the basis of this reaction in the blood. For instance, it is after the patient has failed to breathe for several seconds or a minute or so that the spasm finally relaxes. It relaxes before breathing is established. The sequence is first a tonic spasm, involving the voluntary muscles. We have only an extrusion of air through a narrow glottis and no inspiration taking place. Then we have a relaxation and relaxation precedes the mechanism of breathing, because it is the relaxation of the muscles which are involved in the mechanism of breathing. I question Dr. Murphey's theory of this change. The acidosis, and whatever relaxation takes place, in my mind, must be explained on some other basis.

To employ the method of treatment we should have to have our patients under control with oxygen available to administer.

Dr. N. R. Smith, Tulsa, Okla.—Dr. Murphey's explanation as to how the attack comes about and as to the influence of the oxygen content of the blood do not harmonize with the results of others. Attacks have been stopped by a method exactly opposite to his. I shall be interested to learn whether the repeated application of the treatment has any tendency to reduce the number of seizures. Are we dealing again with a method for symptomatic relief or with something that may permanently reduce the convulsive tendency in a given patient?

Dr. William A. Smith, Atlanta, Ga.—The recent trend of study of epilepsy is along psycho-chemical lines. Several factors have to be considered, such as alkalosis, edema, anoxemia and electrolytic changes in the brain. Thus has developed the treatment of epilepsy by the ketogenic diet, and by the limitation of fluid-intake. Anoxemia is considered by many to be an important factor, and this cerebral anoxemia may be produced by the vascular spasm in the brain which occurs at the beginning of an epileptic seizure. All of these factors are interrelated. Carbon dioxide, as by rebreathing, will stop epileptic seizures, as well as hiccough and other spasms. On the other hand, hyper-ventilation may produce convulsions. In these measures, the acid-base disorder is undoubtedly more important than the concentration of oxygen. We shall never understand epilepsy until we know more of the physiology and metabolism of the brain, and can visualize just what is happening in the brain when a seizure occurs. From the practical standpoint, we seldom see a patient during a convulsion, and what is needed is to know whether oxygen therapy is of value in the prevention of seizures.

Dr. Murphey (closing).—Relaxation occurred after oxygen was inhaled three or four times, or after oxygen was given intravenously in amounts ranging from 60 to 120 c. c. I had one case of status epilepticus reported as L. M., *case 20361*. You will note that it took 240 c. c. of intravenous oxygen to stop the convulsions in this instance.

THE CONDUCT OF LABOR IN PELVIC DEFORMITIES*

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The conduct of labor in pelvic abnormalities has always been one of the hardest problems for the accoucheur to solve. No other complication of labor that confronts the obstetrician has received so varied a treatment nor has any advanced so progressively. A brief review of the subject shows many changes in the last century. One hundred years ago, Meigs, of Philadelphia, wrote:

"Whenever, therefore, a woman has fallen in labor, who is known to have an impracticable pelvis,—if the perforator is to be resorted to, it should be applied as soon as possible, in order that, the child having ceased to exist, all the facilities derivable from incipient decomposition of the foetus may be enjoyed. Twenty-four hours after the death of the foetus, the firmness and cohesion of its soft parts are so much lessened by maceration, in an elevated temperature, equal probably to 99 degrees, that the extraction of the pieces of bone is exceedingly easy. I should, therefore, in such difficult cases, recommend that all attempts to deliver should be delayed, if possible, for twenty-four hours after the perforation of the head."

J. Whitridge Williams, of Johns Hopkins, states that from 1895 to 1905 the operation of version and breech extraction was performed most often at that hospital. From 1905 to 1915 pubiotomy was used chiefly but later discarded.

Other plans of treatment which have been tried out and still have a few followers are: cutting down on the mother's diet during pregnancy with a view of reducing the size of the child. This has not proved successful, in that it did not prevent the child from developing fully and the starvation weakened the mother, making her a poor risk for labor. Then operative induction of labor had its sway and is still used. However, statistics show a greater infant and maternal mortality in the operative inductions than among the same class of patients if they are allowed to go into spontaneous labor at term.

In this paper I shall not take up the subject of pelvic classifications. The diagnosis of the

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absolutely contracted pelvis or the markedly deformed pelvis is so simple that it is usually made by the patient herself and the treatment is obvious.

The types of cases which are most difficult to handle and which I wish to discuss are those with the conjugate vera ranging between 7.5 and 9.5 cm. in length, the generally contracted pelvis, the funnel pelvis, and the flat pelvis, or, in other words, where there is a disproportion between the head and the pelvis.

I shall give two case reports which are typical of those frequently seen in our clinics, and which demonstrate the importance of a careful pelvic examination, including pelvimetry before a patient goes into labor.

Case 54619.—Admitted to the ward October 8, 1929, a colored primipara came in with a history of having been in labor for six days. An unsuccessful attempt at forceps had been made. The fetal heart sounds were good. She had an absolutely contracted pelvis and was delivered by Porro cesarean of a living infant. The mother died from peritonitis a few days later.

Case 54843.—Admitted October 27, 1929, a colored multipara gave a history of one previous forceps delivery of a small child. She had been in labor for twenty-four hours. The fetal heart sounds were good, and the cervix was fully dilated. She had a moderately contracted pelvis with a conjugate vera 8.5 cm. in length. Podalic version was attempted and as the head would not then engage it was found necessary to perform a craniotomy on the after-coming head.

The diagnosis of the moderately contracted pelvis is made by a careful pelvic measurement and by considering the history, stature and race of the individual. The history of previous difficult labor is always indicative of trouble. I make it a rule to view with extreme suspicion any case which has had two difficult deliveries. A history of such conditions in childhood as Pott's disease, rickets, infantile paralysis, or any injury which kept the child in bed during the ossification of the pelvis, thereby preventing it from developing normally, should lead us to suspect trouble.

We are especially concerned, in the South, in the racial differences of pelves. The fact that the colored pelvic measurements are different from the white ones does not seem to be generally understood. Every few months I see colored patients who have returned from the East where they had been delivered by cesarean for a pelvic deformity, and on careful examination I usually find that their pelvic measurements are normal for their race. The colored pelvic measurements found in our clinic in Charleston are about 2 cm. shorter than those of the white race. Parsons, of Los Angeles, reports about the same condition among the Chinese. A comparison of these three races follows:

	White (DeLee)	Colored (Roper Hospital)	Chinese (Parsons)
Sp. 1.	26 cm.	23	22
Cr. 1.	29 cm.	24.50	26
Bi. T.	31 cm.	29	29.31
D. B.	20 cm.	18.50	18.80
C.D.	12.50 cm.	11.40	11.60

The small pelves of the colored and the Chinese races are probably compensated for, by their infants being smaller than the infants of the white race.

Length and Weight in the Newborn

	White	Colored	Chinese
Length	50. cm.	48.6 cm.	50.16 cm.
Weight	3480. gm.	3240. gm.	2958.61 gm.

The x-ray has not given us as much aid in the diagnosis of disproportion as one would think it should. I am sure the future will bring forward some method for studying the pelvis along these lines. We are indebted to Thoms for devising a plan for the x-ray study of the pelvis. However, I fear that it is not practical for most of us at present.

In a case where we believe that there is some pelvic abnormality, the case should be studied carefully for a disproportion between the fetal head and the pelvis. There are several plans of determining whether the head will enter the pelvis. The size of the fetal head *in utero* is determined by measuring the transverse or the oblique diameters of the head and then deducting 1.5 cm. for the abdominal wall. Another method is by grasping the child's head through the abdominal wall, and by placing the fingers against the head, internally. The head is forced down into the inlet of the pelvis. The examining finger then determines whether there is an overriding or whether the head can be forced into the pelvis or not.

These tests are not infallible and the best one that I know of is the test which is brought about by the natural forces of the uterus in labor.

While statistics show that the majority of borderline cases will deliver spontaneously if left to nature, the now generally adopted method of handling these cases is by trial labor and so far this has proven to be the best. This treatment is to allow the patient to remain in labor for four hours, using every precaution not to infect the birth canal. If, after suitable time, the head has not engaged, the patient may then be de-

livered by laparotrachelotomy. There is considerable discussion as to how long we should permit a patient to remain in labor before considering the trial a failure. Williams, discussing a paper on this subject, said:

"We cannot speak of a trial labor until the cervix is fully dilated, or until we have had an opportunity to observe whether the uterine contractions can force the head past the obstruction or not. Consequently, to fix any number of hours, and to say that after their expiration the woman has had a trial of labor, leads to false conclusions, for in my estimation, we are not justified in using the term until the patient has passed into the second stage."

DeLee, discussing the same paper, said: "I think a test of labor is the way a woman goes into labor. A test of cervical dystocia is just as much a test of labor as a test of mechanical pelvic dystocia. A woman goes into labor, for example, with a tight, hard, long, uneffaced cervix. If, after eight or ten hours, the pains have not affected this appearance, I should call it a test of labor."

In my opinion an experienced obstetrician can prognosticate fairly accurately what is going to be the final termination of a case after it has been in hard labor for four to six hours. Harris and Williams, by examining cultures taken from the uterus when performing abdominal sections, found that the uterus is not infected until after four to six hours of labor. It would then seem that where trial of labor had not brought about engagement, delivery by the low cesarean would be the method of choice.

There are many cases that one sees in consultation after the patient is infected, or when one has failed properly to estimate a pelvic deformity in which forceps or podalic version may become necessary, when the natural forces are unable to complete delivery. The use of forceps, unless the head has engaged, has been pretty well discontinued, for if nature is unable to bring about engagement certainly we cannot do it with forceps. Podalic version will sometimes be necessary and in this instance I have found it very valuable to tie the hands with a piece of tape before bringing the breech down. To do this one hand is brought down as low as possible, then with a piece of strong, narrow, sterilized tape the arm is tied by making a slip-knot at the end of the tape and passing this over one's finger and inserting the loop into the birth canal and over the hand which has been previously brought down. Traction on the free end of the tape fastens the knot around the wrist, and the other hand is brought down and tied in the same manner. The hands are then replaced into the uterus and the version performed. The arms are brought down by making traction on the tape as the body of the child is being delivered. This saves much delay and injury to the fetus and will give one more time for applying forceps to the after-coming head if that should become necessary. Potter's method of delivery of the after-coming head is certainly very valuable in these cases. He directs pressure against the head from above the symphysis instead of using the obsolete Moriceau method. If the case is to be delivered by the natural route one should allow labor considerable time, and after dilatation is complete, rupture the membrane to aid engagement and molding of the head. A disproportion between the head and the pelvis means a long, tedious labor. Unfortunately, there are many abnormal presentations in pelvic deformities. They should be diagnosed early and converted if possible. However, if the pelvis is small, the patient should be delivered by cesarean.

Drug induction of labor two weeks before the date of confinement is worth trying. It is successful in a small percentage of cases.

CONCLUSIONS

(1) I wish to emphasize the importance of a careful pelvic examination before the beginning of labor.

(2) All borderline cases should be hospitalized for labor, and given a thorough trial before operative interference.

(3) We should bear in mind the racial differences between pelves.

(4) Much delay in versions or breech extraction can be prevented by tying the arms and bringing them down with the body.

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DISCUSSION *(Abstract)*

Dr. J. Bay Jacobs, Washington, D. C.—We are generally taught that in order to establish the length of the true conjugate diameter, one must subtract 1.5 to 2 cm. from the diagonal conjugate, depending on the height and inclination of the symphysis. I think this should be modified to some extent, because the amount to be deducted depends not only on the height and inclination of the symphysis, but also on the distance

of the sacral promontory from the pubis, and the station of the former as compared to the lower border of the latter. To my mind, the angle included between the symphysis and the conjugata diagonalis is the most important factor in determining the length of the true conjugate. I have found with the inclinometer that the size of this *obstetric angle* varies in all patients, and for that reason the common rule of subtracting 1.5 to 2 cm. from the diagonal to determine the length of the true conjugate, often does not apply.

Then, again, it is interesting to note that the cephalic measurements of the fetus do not vary in direct proportion to the weight. Thus the skull of a heavy baby often has diameters of normal length or only slightly over. Even in a primipara where a head overrides, it may take several hours for engagement to occur. Just recently I had such a patient with a large pelvis and a large fetus, and she caused me much concern, because the head did not enter the brim until the second stage of labor. Normal delivery followed. What may be considered a real test of labor, varies with each individual case. To follow a general rule may interfere with good obstetric judgment.

The idea expressed by Dr. Wilson of putting a fillet on the baby's hands in order to prevent the arms from going over the head, I think is excellent. As many of our versions and extractions are performed late in labor and under adverse circumstances, this common complication can thus be avoided.

Dr. James R. McCord, Atlanta, Ga.—I cannot tell, unless I have an absolutely contracted pelvis, whether the fetus can be delivered or not. I cannot tell by the overriding head whether it will come through the pelvis, but I know that about 80 per cent will come through regardless of what we think about it. We have a clinic in Atlanta in which we deliver approximately 1,500 women a year. They are all negroes. It is the only haven of refuge for the negro population of 75,000 persons. All of our normal cases are delivered by senior students. We have 25 beds, three beds in each ward. Twenty-six per cent of the admissions into the hospital have been obstetric cases, and in the last 9,000 cases we have done something like seventeen or eighteen cesarean sections. In 1,500 cases we had only 45 forceps deliveries. We also have a syphilitic incidence of 15 to 20 per cent, and chronic nephritis, 5 to 10 per cent.

You cannot put a time limit on obstetrics. We should teach coming obstetricians the mechanism of labor, and that every woman in a given time will have her baby, or certainly get the baby in position where it can be easily delivered. As long as the pulse is all right and the uterus is relaxed between pains, regardless of whether we have been fifteen minutes or fifteen days, we have absolutely no indications for interference.

Dr. Willard R. Cooke, Galveston, Texas.—Pelvimetry is a difficult art. If you have ever checked back over a large number of readmission cases in your hospital, you will probably have reached the conclusion, as I did, that the pelvis is not made of bone, but of cardiac muscle; and that the difference between the systole and the diastole is sometimes as great as 1¼ inches. Even if the measurements are accurately determined, they do not tell the whole story. The essential thing is disproportion; whether this be due to an oversized head or an undersized pelvis. Another important factor in a case of moderate disproportion is the character and efficiency of expulsive forces. There are two things which we ought to determine definitely before a patient goes into labor. Early in pregnancy the inside of the pelvis should be measured to see whether there is any tumor or bony deformity, or whether the patient has a funnel pelvis. It is difficult to measure the transverse diameter of the outlet accurately. I have found a very simple "rule of thumb" which is of great service to me. If you know the diameter of the closed fist at the knuckles, and if the knuckles will pass between the tuberosities of the ischium a baby's head of similar size will have room for exit. Or, another rule in managing cases of moderate disproportion is to make weekly observations of the relative size of the head and of the inlet by impression. If there is evidence of moderate disproportion two weeks before term, then try to determine whether it would be better to induce labor or to allow the patient to go to term for trial at labor, to be followed by section if necessary, or to decide at once in favor of section. In cases where disproportion becomes evident before the thirty-eighth week, section under ideal circumstances at term is probably the preferable mode of delivery; but in doubtful cases it should always be preceded by a test of labor. There are two things which can never be foreseen when the decision to induce labor is made:

(1) Whether the cervix will dilate.

(2) Whether the patient will respond to induction by adequate pains; and with the rules accepted at present in regard to cesarean section, once labor has been induced, cesarean section is no longer to be employed in case of failure of the induction.

There is much difference of opinion in regard to the true status of the test of labor. One is often misled if the primipara becomes excited from inadequate pains which accomplish nothing and do not constitute the test of labor. Such a patient will usually respond beautifully to ⅛ to ¼ gr. morphine given when she becomes moderately exhausted. As soon as the effect of the morphine wears off most patients will begin to have adequate pains and the rest of the course of labor is normal.

Our own conception of the test of labor is one in which the pains are frequent, forcible and effective to the extent of producing the normal rate of dilatation of the cervix without accomplishing any descent of the head. Such a test of labor should not be allowed to continue more than four, or at the outside, six hours, if cesarean section is to be resorted to.

Dr. Mary Freeman, Perrine, Fla.—In seventeen years of practice I have encountered only three cases that demanded cesarean section. One was a ruptured uterus, another an ossified cervix and the other, a right occipito-posterior, a large child and small mother.

I always give nature a chance and interfere only when the patient shows inability to deliver. A few times it has looked as though the diameters were too small to deliver, but after nature softens all the joints, the bony canal seems to expand.

Dr. E. L. King, New Orleans, La.—We all believe in pelvic measurements and we teach them, and I am firmly convinced that every patient should be measured, but that does not tell the whole story. We must consider in each case, as I try to tell my students, whether this particular baby will pass through this particular pelvis. A baby that weighs six pounds will pass through a pelvis where one that weighs eight pounds will not. That is perfectly obvious, but is sometimes forgotten.

An early vaginal examination should help us to determine the character of the pelvis. In my private work I try to estimate something about the interior of the pelvis when I make my first vaginal examination, and then the pelvic measurements give me some information about the exterior. Besides that we have to consider in the borderline cases whether a particular baby can go through this particular pelvis, and that is hard to determine until near the end of pregnancy.

The best test is the test of labor: four to six hours of real pains. Some patients with mild first stage pains will show little if any dilatation. In such a case it is frequently wise to give a dose of morphine, let the patient rest and then try again, and we are surprised to see that strong pains develop, the head engages, and delivery is completed with little or no difficulty.

It is a question in my mind sometimes whether it is a triumph of obstetrics when we do manage to get a baby through a small pelvis, fight through a difficult forceps delivery with the associated intracranial hemorrhage, and finally save the baby, but what is its fate later? Some are good citizens, and some we have seen around the streets and in clinics, little more than vegetables. It is not always greatly to our credit that we deliver a live baby, unless it is mentally and physically all right. Sometimes it would be better judgment to do a cesarean section than run the risk of getting this type of infant, dead or worse, particularly if there is a history of previous difficult labor, or a dead baby.

Dr. McCord brings out a point that is well worth noting, and that is that we do not seem to have as much trouble with negroes as with whites. Is that because we let the negroes labor longer (they are not private patients), or because there is really some racial difference? I notice in the wards of the Charity Hospital, where we have a white service and a negro service in two separate wards, that the incidence of operative obstetrics is lower in the negro service than in the white. Interns prefer the white service because there is more operative work. It may be because the Charity Hospital is almost the only haven of refuge for the colored race in New Orleans, and they all come there, normal as well as abnormal; whereas in the white service the women are frequently patients who have been unable to deliver themselves outside the institution in the hands of midwives or physicians. Most of the normal obstetrical work on the negro is done in Charity Hospital; whereas most of the normal work on the whites is done at home, and the difficult cases are sent to the hospital.

Dr. S. H. Starr, Louisville, Ky.—We think less of the pelvic measurements than of the actual proportion of the pelvis to the size of the baby's head. There are five factors concerned in determining whether a given fetus will deliver through a given pelvis: the size of the pelvis, the size of the head, the position of the fetus, the force of the contractions and the malleability of the fetal head. Post mature babies have not only larger but also harder heads.

Many times we see cases of dystocia which are supposedly due to difficulty in dilatation of the cervix. Frequently this is due to the fact that there is a disproportion and the head does not descend low enough to press against the cervix and produce dilatation.

Dr. W. B. Anderson, Nashville, Tenn.—I recently saw a patient who had had two cesarean sections because of contracted pelvis. While she was changing her residence from one town to another, labor started, and she gave birth to the baby before she got to the delivery room.

A few months ago in the clinic at Vanderbilt some students brought in a patient and said she would have to have a cesarean, that they had given her the test of labor. Upon examination the fetus was found in a posterior position, the head riding on the brim. You might say that was student work, but it also happened a few months ago that a patient was brought in thirty miles to the hospital for a cesarean section. A deficient antero-posterior diameter had been diagnosed. She was another case of occipito-posterior, and when version was done she delivered very nicely.

Dr. M. C. Wilson, Miami, Fla.—I want to ask Dr. Wilson in his closing remarks to tell me what he thinks of the x-ray in this matter. Personally, I do not depend a great deal on it, but I think it should be more useful.

In Miami, among about 1,000 patients in four years, I have not seen a contracted pelvis. I wonder whether we have a better class of patients, reared in better homes, who have no contracted pelves. I have almost come to the conclusion that there is not a white American-born woman in Miami who cannot be delivered normally if we give her time. Many cesareans are done, but I think it is because the physician has not confidence in his judgment to determine a fairly normal pelvis, or else he is afraid of the censure of prolonged labor. Some of the easiest labors have been long labors. I wish to commend the remarks of Dr. McCord about the length of labor. That is entirely dependent upon the woman's condition. If she is quiet, is not vomiting, has no elevation of temperature, and the pulse is not too fast, the blood pressure is not down, and she is able to take some nourishment, she can be in so-called labor for days and still she and the child will be in good shape.

Another very good point that Dr. Wilson brought out is the crux of the whole matter. If you are sure of your position, wait until the cervix is dilated. If you have full dilatation of the cervix and the head does not come down you may start something; but while you are waiting for the cervix to dilate be sure you have not an occipito-posterior position, although that is not always easy. When I am in doubt on that point after palpation I do not hesitate to give the patient a little chloroform or gas to relax her, and make a thorough examination. If I have an occipito-posterior and the woman has been in labor, I do not hesitate to introduce my hand a little further, and with two fingers over the nearest arm, flap the baby over on its belly. You are apt to keep the position in that way. I think we make a mistake in waiting too long, until the woman has exhausted her expulsive powers, before we correct the posterior position. Some may consider it radical to put the hand into the uterus; but under modern obstetrical technic it can often be done and you will have no further difficulty in rotation.

Dr. Wilson (closing).—The best method for studying the pelvis by x-ray was described by Herbert Thoms. He uses a lead plate with perforations exactly 1 cm. apart. The picture of the pelvis is taken with the plate a certain distance from the pelvis; by counting the perforations the exact size of the pelvis can be ascertained. This

method is described fully in the *Journal of the American Medical Association,* May 4, 1929.

Gross disproportions between the pelvis and the fetal head, exostosis and marked deformities are easily demonstrated by the x-ray.

TREE POLLEN HAY FEVER AND ASTHMA IN THE SOUTH*

By I. S. KAHN, M.D.,
and
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Pollen plates run through the year, in connection with local botanical surveys, give us an extremely accurate method of determining from day to day the variety and comparative amounts of pollen in the atmosphere. The actual differential identification of these various air borne pollen granules so detected is ordinarily not a matter of any great technical difficulty, and the usual confirmatory diagnostic skin tests are as easily secured with the causal tree pollens as with the corresponding pollens of ordinary late spring and early fall hay fever, our usual types.

The tree pollen causing the largest amount of distress in any of the Southern states is that of the mountain cedar *(Sabina* or *Juniperus sabinoides).* Its annual victims can be numbered by the thousand. However, as this growth is restricted to Texas and further west, it is of little or no interest to the South in general, and is not being given any further mention in this article, which takes up the consideration only of such tree growths as are common to all the Southern states. The pollination season of this tree, from late December to early February, distinctly antedates all the other tree seasons, so that the diagnosis error of inclusion of this mountain cedar factor is practically impossible. All the trees considered herein pollinate for several weeks between late February and early May.

The figures quoted in this paper are from our 1929 private office practice, and since the onset and cessation of the pollination seasons of the various trees common to the South to be mentioned differ only by a few days in various localities, they probably give a very good cross section of conditions that actually must exist throughout the entire region.

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Incidence.—(1) Pure tree pollen hay fever cases restricted to tree pollination season only. Of these, we have seen three cases only this year, and the comparative rarity of such cases is observed by others. Of uncomplicated spring tree pollen asthmas, we saw no new cases this year and have seen only one or two in past years. It is this very infrequency of occurrence of uncomplicated tree pollen hay fever and asthma that leads to its overlooking as a complication of hay fever and asthma due to other pollen causes.

(2) Tree pollen hay fever complicating hay fever of other origin. These cases invariably date their symptoms from one to several weeks before the regular pollination season of the spring grasses, and if the etiologies of all such presumed grass hay fever cases dating their onset previous to April 15 are gone into, they will fall into this group. In other parts of the South, with the exception of Florida, where grass pollination begins in January, symptoms starting earlier than April 15 should call for the investigation of sensitivity to tree pollens. We saw 21 positive cases of this group this year.

(3) Tree pollen asthma, complicating asthma, due to other pollens. In past years, we have noted in our grass or grass and ragweed asthmatics, decided relapses in March and early April, especially in middle or late March. Inasmuch as at this time most of these cases, as well as the spring hay fever cases, are undergoing preseasonal grass desensitization treatment, we formerly ascribed these relapses to overtreatment, nonspecific weather conditions or to the pollen of one grass, *Lolium perenne,* whose pollination season antedates that of the early important spring grasses. It was only when these cases were retested for the tree pollen of the air present at such times, as detected by pollen plates, that the true state of affairs was revealed. We had 23 of these cases this year.

A cause of the previous failure of such etiological diagnoses in many of these cases, unsuccessfully handled in preceding years, is the fact that in our experience, for some reason or other, tests for our local tree pollens, routine in our office for several years, have frequently been entirely negative until the onset of clinical tree pollen symptoms. Tests then repeated showed positives, and relief was secured by proper specific treatment in a gratifyingly high percentage of the cases.

Diagnosis.—Tree pollen extracts, on the whole, are highly antigenic, and tests intradermally

done during the pollination season had better be started with extracts of 1:50,000 dilution in order to avoid unpleasant shock reactions. Dermal tests, for the convenience of safely permitting the application of several tests at a sitting, we have found best done with previously prepared 1:50 extracts, removing the extract from the sites tested as soon as there is the slightest indication of commencing wheal formation. In our experience also, tree pollen is highly specific, there being no common sensitivity even to closely related species of the oaks. We differ here from the findings of Dr. Scheppegrell.[1] Live oak has been our local oak factor, the other oaks of our community often giving negative skin tests in our positive live oak cases.

Tree pollen sensitiveness is usually multiple, several botanically non-related groups being incriminated in one individual. Curiously enough, however, our uncomplicated pure tree hay fever cases were clinically sensitive to one tree and one only.

Also, as a result of our studies with pollen plates, tests and clinical observation, the duration of tree pollination hay fever and asthma seasons is much closer to two months than to the three weeks' period usually mentioned. For instance, pollen plates showed that live oak in San Antonio in 1929 ran from March 9 to May 6, and pecan, from March 30 to May 9. Cottonwood pollinated from February 27 to April 14, all with corresponding coincident clinical suffering.

To make the positive skin tests of clinical value, symptoms must, of course, coincide with the presence in the air coincidentally of such pollen, and cease with its abeyance or after specific treatment. As with other pollen tests,[2] in tree pollen hay fever and asthma, skin tests are at times either insignificant, diagnostically inconclusive or absolutely negative.

Treatment and Results.—Preseasonal hay fever treatment in our few cases has been almost perfect. Seasonal treatment in the hay fever cases by the intradermal method of Phillips[3] has given very satisfactory results, two or three treatments often clearing these cases for the entire season. In asthma, the results have been excellent, but not so prompt or satisfactory as in the hay fever cases. The proper doses here seem more difficult to determine, more numerous doses are required, and overdosage at times is rather hard to avoid. However, this method certainly in our hands has proven of decided efficacy, and has given us a means of securing entire or almost complete relief within a few days in the large majority of our cases.

Botany.—The only even partially comprehensive report on this subject of tree pollen hay fever in the South is that of the late Dr. Scheppegrell,[4] and in this paper, the relation of tree pollen to bronchial asthma is not mentioned.

Obviously, one writer cannot cover this field completely. New tree pollen causes will come to light from year to year as more attention is given to this point by pollen workers in various localities, some causes not yet reported, some only suspected and not yet definitely proven. All we can offer at the present writing is our own experience, enumerating also such causal tree growths as we have been able to gather from the reports of other workers. In some sections of the South, such as Florida, with a more or less distinctive vegetation, no studies of this kind whatsoever have been reported.

The trees of San Antonio found incriminated in this study, many common to the entire South, are ash, cottonwood, willow, oak, hackberry, pecan, box elder, and sycamore. Dr. J. H. Black,[5] of Dallas, adds elm as one of his common offenders. Our heaviest local hay fever producers have been pecan and live oak. We have seen one or two cases of hay fever, due to close contact with mesquite, an insect pollinated tree. It, of course, is a Western and not a Southern growth. Its only interest in this connection lies in the possibility of similar trouble from botanically related Southern trees of the mimosa family.

Other trees mentioned as causes of hay fever growing in the South are walnut, hickory, sweet gum, maple and birch, none of which grows in San Antonio, and of which we have no direct knowledge. Dr. Scheppegrell[6] mentions ligustrum, which so far we have been unable to confirm. This may be due to the fact that in San Antonio this tree ordinarily is used as a small ornamental tree and is not permitted full growth as a shade tree, being either trimmed back just before blooming or cut as a hedge. Dr. Bernton[7] last year added paper mulberry. Obviously, anyone investigating this subject will have to be familiar with the tree growths of his particular community. Most of the trees mentioned give off enormous amounts of pollen, especially live oak and pecan, are not insect pollinated, and there is no reason why they should not cause trouble to susceptible individuals. The fact that we have seen 56 cases of this type in one season

shows their comparative abundance and the importance of this subject.

CONCLUSIONS

(1) Tree pollen hay fever and asthma are of decidedly higher incidence in the South than is generally recognized.

(2) Many cases of asthma owe their early spring relapses to this source.

(3) The causes are highly specific.

(4) Relief is prompt, and in almost all the cases offers very little difficulty.

*Since this article was written, personal investigation of the botany of northern Florida in November, 1929, showed an abundant cedar growth identified as *Sabina virginiana* and *Sabina barbadensis* (the latter commonly called Cuban cedar) by Professor M. D. Cody, of the Botany Department of the University of Florida, closely resembling *Sabina sabinoides*, the Texas mountain cedar mentioned. Pollen plates set out in St. Augustine and mailed in by Dr. Herbert E. White, of that city, in early February, 1930, showed abundant cedar air pollination. Winter hay fever and asthma do exist in Florida (Dr. T. Z. Cason, of Jacksonville, personal communication). It is probable, but not yet definitely proven, that cedar is a factor in late winter hay fever in Florida. Weekly pollen plates sent in by Dr. White since November, 1929, did not show this pollen before February. According to Small's "Flora of the Southeastern United States," the growth of *Sabina virginiana* extends throughout temperate North America, while *Sabina barbadensis* is found on the coastal plain from South Carolina to Florida and Texas, and also in the West Indies.

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DISCUSSION (*Abstract*)

Dr. John P. Henry, Memphis, Tenn.—From what Dr. Kahn has just said, it is obvious that each of us must be familiar with the botanical flora of the territory in which we work. We have made a survey over, roughly, 200 miles of Memphis.

Probably the most abundant tree in this territory, considering both the cities and the country at large, is the oak. In the city of Memphis, however, there are many elms and these are the first trees to pollinate, beginning about February 7. It is quite an advantage to know the pollinating season of the various trees, for sometimes, in the absence of definite skin tests, one can select proper pollen extracts for treatment on history alone.

After the elms (*Ulnus alata* and *americana*), the following trees and a few of the early grasses begin to pollinate in about the order named: hazel-nut (*Corylus americana*), red maple (*Acer rubrum*), cypress (*Taxodium distichum*), annual bluegrass (*Poa annua*), cottonwood (*Populus deltoides*), red cedar (*Juniperus virginiana*), trembling aspen (*Populus tremuloides*), black poplar (*Populus nigra*), Lombardy poplar (*Populus italica*), box elder (*Acer negundo*), river birch (*Betula nigra*), hornbeam (*Carpinus caroliniana*), red oak (*Quercus rubra*), ironwood (*Ostrya virginiana*), yellow pine (*Pinus echinata*), willows (*Salix spp.*), Southern hackberry (*Celtis mississippiensis*), sweet gum (*Liquidambar styraciflua*), white ash (*Fraxinus americana*), pig-nut hickory (*Hicoria glabra*), shagbark hickory (*Hicoria ovata*), marsh foxtail grass (*Alopecurus geniculatus*), American beech (*Fagus grandifolia*), button-ball, sycamore (*Platanus occidentalis*), post oak (*Quercus stellata*), white oak (*Quercus alba*), pecan (*Hicoria pecan*), red mulberry (*Morus rubra*), little barley grass (*Hordeum pusillum*), Kentucky bluegrass (*Poa pratensis*), black or sour gum (*Nyssa sylvatica*), black walnut (*Juglans nigra*), curled, narrow dock (*Rumex crispus*).

Dr. Narcisse Thiberge, New Orleans, La.—Tree pollen sensitization has greatly interested us. Of the 33 varieties of trees which grow in Louisiana, over half of them, representing seven families, have been proven to cause or aggravate hay fever.

We can confirm Dr. Kahn's experience in the active reaction produced by tree pollen extract: we noticed that this sensitivity accompanied and almost corresponded to grass sensitization and not ragweed. By making control tests with saline, we have not found it necessary to use a weaker solution than the usual 100 units to the c. c.

Though we can recall many cases confirming Dr. Kahn's study of the role played by trees in hay fever and asthma, one case of single elm pollen sensitization stands out most clearly: coming, increasing and disappearing with the tree's bloom. The elm in the South is practically the first tree to bloom, though the willow, aspen and maple are almost as early. The Hay Fever Prevention Association Laboratory in 1917 received an inquiry about the part played by the cedar.

In individual cases of sensitization when the tree is suspected:

(a) The nasal discharge should contain the pollen.

(b) The exposed plates should show them.

(c) The intradermal test should be positive, and,

(d) The attack should occur during the pollinating season of the tree accused.

Proximity to the tree is not necessary, as the pollen has been found 15 miles away and 6,000 feet in the air.

The interesting feature about tree pollen is the height from which it takes the air. In the same wind, a pollen 10 microns, which goes 184 feet, when the plant is 2 feet, will go 268 feet when the plant reaches 5 feet. How much farther a tree pollen of similar size would travel, you can readily see. Increase that wind from 2 miles to 30 and you can readily see how much farther it will travel.

We have not found it so easy a matter to identify the pollen as Dr. Kahn indicates, but it is well to remember that the willow, lygustrum, box elder, oak and sweet gum pollens are oblong, the mulberry and cedar odd-shaped and the balance of the hay fever tree pollens globular. Of the oblong, the oak is the smallest;

of the globular pollens, the mimosa 6 microns, and the oak, 20 microns, are the smallest. The others are quite large.

The difference of opinion about the ligustrum and many ornamental trees comes from the fact that here the pollen, being larger, does not travel far. A pollen 10 microns will travel 180 feet in a 2 miles per hour wind, while one of 50 microns will travel 2 feet.

We have prepared photomicrographs of the trees mentioned by Dr. Kahn, with the size of the pollen indicated. By studying the size and the solubility of the surrounding envelop, their reactive danger may be ascertained. The finding of the pollen on the plate or in the nasal discharge is not necessarily confirmatory, as some of these pollens have a tough, or a resinous envelop which prevents absorption. Also, an interesting point has occurred to us: some pollens retain their fertilizing power over a long period, while others lose it promptly. Grasses lose their fertilizing power in two days, the willow in one day, ragweed in six days and trees from seven to seventy-seven days; the mimosa in seven, and pines in sixty days (but as these last have a resinous envelop, they are more slowly absorbed and do no damage). The pollen sensitizing power may bear a direct relation to the longevity of the pollen grain.

Dr. Ray M. Balyeat, Oklahoma City, Okla.—There has been a tendency in the last few years for much attention to be paid to grass and to ragweed, and to thistle, and so forth; and they are important, of course; but I think probably too little attention has been given to tree pollen. In the first place, it has not been given a great deal of thought because of the fact that all of us feel that the period over which the trees pollinate is very short; and frequently that is true. However, one must stop and consider the fact that, although the period over which some trees pollinate is short (and we will exclude the mountain cedar, because the period over which it pollinates is long), yet a great many people are sensitive to a number of trees. For instance, in Oklahoma City the elm tree begins to pollinate about March 6, as a rule, and although we have rather bitter winters and may have two or three spells of zero weather in March, yet in some years March is a mild month and we may have a period of twenty days or more without frost, and then the elm is a considerable factor. The cottonwoods begin to pollinate in April. Some people may be sensitive to oak, and the oak begins to pollinate about April 10. It is not unusual to find patients who are sensitive to elm, oak and cottonwood. The elm starts their symptoms in the fore part of March, the cottonwood adds to their symptoms about April 1, then from April 10-15, the oak begins to play a part. Such individuals, therefore, would have the mucous membranes of the nose or bronchial tree irritated, off and on, from the early part of March to May 1. In such a case the period over which tree pollen would irritate the mucous membrane would be as long as the period over which the ragweed would irritate the mucous membrane of one sensitive to ragweed. During the winter and spring the chilly changeable weather would also intensify the irritation of the pollen.

Then we must take into consideration the fact that trees may be a so-called secondary factor. For instance, I may have a patient sensitive to cottonwood. The cottonwood season lasts just two weeks, so I may suggest to the patient that he do without desensitization for cottonwood and be desensitized for grass. But unless I desensitize him for cottonwood and let him go into the grass season with a perfectly normal nose he will suffer. The oak is of tremendous importance, and unless we let patients go through the oak period comfortably and go into the grass season with a normal nose, good results cannot be hoped for from the grass treatment.

We find many of our tree pollen patients who suffer from hay fever, who have associated with their hay fever mild to severe asthma. Both oak and elm are very toxic. If you allow the pollen to irritate the bronchial tree it is going to make it much harder for them during the summer.

Our results in desensitizing to tree pollen have been fair, but not as good as reported by Dr. Kahn. The most severe cases of seasonal asthma that we see come from oak, and it is very difficult to give them complete relief by desensitizing. If we obtain 80 or 90 per cent relief we feel that we have done very good work.

Dr. H. M. Davison, Atlanta, Ga.—We have found in Atlanta, also, that while there are very few tree-pollen cases, comparatively, yet the cases that we have are very strongly sensitized, and they have more asthma than the other hay fever cases do. Also, we have comparatively more cases of asthma without hay fever from the tree pollen than we have from the grass pollen or the ragweed pollen. This last year I have made some pollen counts in Atlanta. We exposed our slides on the sides of houses and on window ledges, whereas the proper way, I understand now, is to expose them so that the wind may hit them unimpeded from any direction. The first pollen that appeared was tang alder, the next was elm, then cottonwood, then pine, then ash, then oak on March 17, hickory on April 10, and a little later, walnut. The time that these pollens left there was also rather interesting. The alder starts about March 2, the elm March 9. Pine continues to the last of July sometimes, though, of course, it usually stops much earlier. The remainder of the trees had a marked drop the last of April.

My experience in testing for oak pollen varies little from Dr. Kahn's in that practically all patients who react to oak at all react to most of these pollens. Live oak and post oak gave the strongest reactions. I found that patients who react to hickory and oak react to chestnut and pecan. We have not many pecans in Atlanta, but in south Georgia the country is practically covered with them, and it has become a large factor in the production of hay fever in our last few cases. We have not been successful in the treatment of these cases. I do not know whether the air is too full of pollen or whether we give them too large doses. Practically all of them have asthma with the hay fever. I should like some help from Dr. Kahn on this point.

The state of Georgia has cooperated with the state of Alabama in making a list of the trees and bushes by number. I am now having some slides made to show this. The physician who treats hay fever should have a chart of this kind and have pollen plates made by the individual patient to find out the cause of his trouble. There must be an absolutely local effect from trees that grow in large numbers in any one place. As Dr. Thiberge said, the size of the pollen must be an enormous factor in the production of this disease.

Dr. Kahn (closing).—We have always worked on the basis that the tree pollen factor is negligible, because a pure tree pollen hay fever case is not common. Last year we saw three cases only. But as complicating factors of other hay fever, it is decidedly important,

especially from the point of view which Dr. Balyeat mentioned. If the patient goes into the grass hay fever season with his nose wet and blocked from tree hay fever, the results from treatment for the grass hay fever are apt to be poor.

The point Dr. Thiberge brought out, about the distance these pollens blow, is very interesting also. I had a druggist last year who lived quite a distance out of the country where we have cedar. This man developed winter hay fever. I tested him and decided that he was a typical cedar case. He said that was impossible; they did not have cedar in his country. I gave him some pollen plates, and although the only cedar growth was ten miles distant, we found the pollen on the plates.

The question of tree pollen has to be a local affair. A particular tree may pollinate two or three months in my community, but I cannot say that Dr. Davison is not just as correct when he says it pollinates only two or three weeks in his section.

I am glad Dr. Thiberge spoke of the tremendous sensitivity to these tree pollens. I have had two or three very unpleasant reactions from tree pollen tests.

Why some pollens give positive skin tests only at the time that the patients are having hay fever from them, not when they are clear, I do not know. We see many patients with grass cases of asthma and hay fever, and many ragweed cases, whose skin tests are absolutely negative, and trees parallel along the same line. The sensitivity of a patient cannot invariably be gauged by the skin test. It is a practical method, often correct, often incorrect.

INDICATIONS FOR THE USE OF RADIUM IN GYNECOLOGY*

By GERRY R. HOLDEN, M.D.,
Jacksonville, Fla.

Radium has been for many years firmly established as a most valuable therapeutic aid in gynecology. Nevertheless, absolute indications for its employment are not as yet entirely crystallized, and possibly never will be.

Many general principles governing its use have been more or less universally accepted and, as time goes on, we shall, no doubt, have a greater elaboration of these principles, and a more specific application of them in individual cases. But a certain latitude will always be allowed in using them. The judgment of the gynecologist must play a great part in their application; a greater part, possibly, than is allowable in deciding similar questions in the field of operative gynecology.

This paper does not purport to be an exhaustive discussion of its subject. It represents, rather, certain personal conclusions, based on a study of the literature and colored and modified by the experiences and the observations incident to several years' employment of this therapy.

Of the many pathological conditions in gynecology, radium is of actual value in only a few. The large majority of radium treatments are given for one of three conditions: uterine cancer, myoma, and essential hemorrhage. Our first consideration will be the indications for treatment in each of these three conditions.

Cervical Cancer.—The status of radium in the treatment of early cervical cancer has altered greatly during the last few years, indeed during the last few months. Whereas, formerly conservatism declared operation to be the treatment of election, we have today ample justification should we make radiotherapy the method of our choice. For a number of years many statistical reports have appeared comparing the end results of early cervical cancers treated by radium with the end results of those treated by operation. The general trend of these reports has seemed most definitely to indicate that there is little difference between the final results obtained in either of these two methods.

Finally, the report of the committee on the treatment of malignant diseases with radium and x-ray, appointed by the American College of Surgeons, as given in *Surgery, Gynecology and Obstetrics* of August, this year, states: "The choice between operation and radium in the treatment of early and favorable cases of cancer of the cervix is an open one."

Of course this by no means relegates early cervical cancer exclusively to the radium field. A skilled operator will surely get better results than will a sorry radiologist. But the converse is also true. Moreover, it does justify the faith of those of us who for years have felt that we were getting just as good results by using radium as we formerly obtained by extensive operations.

The treatment of inoperable cervical cancer has been for a long time entirely by radiation; but a routine which uses radium treatment in every inoperable cervical cancer, regardless of the extent of growth or of the general condition of the patient, is unjustifiable. It is true that frequently the results of radiation are much better than we should expect, judging from the original condition of the patient at the time of treatment. Such occasional happy incidents do not justify radiation when marked cancerous cachexia and general metastases are manifestly present; when massive involvement of vaginal walls is such that

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rectal and vaginal fistulae will be among the other distressing post-treatment sequelae. Massive radium treatment in terminal cases does no good, often makes the patient worse, and brings discredit upon the surgeon and upon his profession. A small dosage of radium, intended to act as a palliative, gives results which are no better, oftentimes not so good, as those which may be obtained with some of the palliative treatments which were used in the pre-radium days.

The treatment of carcinoma of the fundus, for reasons which are doubtless familiar to you all, is generally considered to be surgical, unless operation be contraindicated by local extension or general condition. The excellent results which are frequently obtained in such cases as are radiated seem to suggest, however, that possibly early cases may some day be considered in the same class as early cases of the cervix.

Myoma.—Probably the majority of all myomatous uteri should be handled surgically. But a large minority is amenable to radium treatment. Certain rules have been formulated for our guidance in deciding which treatment should be employed in a given case. Some of these rather generally accepted rules are much better than others. The least valuable is that standard which judges by the gross size of the tumor, whether it should, or should not, be radiated. Of far more value is the general formation of the mass, especially with relation to the uterine canal. A fairly symmetrically developed myomatous uterus, with the uterine canal so situated that the whole growth can be fairly uniformly radiated by intrauterine application, can often be successfully treated, although it may be much larger than the size of a four or five months pregnancy. On the other hand, an asymmetrical tumor, developing wholly from one side of the uterus, with the uterine canal pushed away to one side of the growth, cannot be uniformly treated by intrauterine radiation, even though the gross mass be much smaller than that of the first example.

Pedunculated and submucous myomata are generally regarded as not suitable for radiation. That is certainly true in infected submucous growths. Many such growths, not infected, can be satisfactorily handled if care is used. In the case of pedunculated nodules certainly no one would think of radiating the typical large, multinodular, myomatous uterus. Small subserous nodules, however, need not necessarily debar one from radiation. They usually yield easily and without complication to the requisite dosage.

The question of age, in selecting our patients for radiation, is of great importance to the conscientious gynecologist. For good results in myomata it is necessary to give such a dosage that the menopause will necessarily ensue. Certainly in younger women such a course should not be chosen unless operation is for some reason ill advised. Supravaginal hysterectomy, with preservation of the ovarian function, is absolutely the procedure of election in younger women.

The next consideration, one with which probably all would agree, is that the diagnosis of myoma must be absolute, and that the growth must be uncomplicated. Should there be any doubt about the diagnosis, should there be any possibility of tubal or ovarian complications, the case is without question surgical. It is, of course, perfectly possible to radiate in the presence of an old chronic pelvic inflammatory disease without stirring up the old inflammation. But the danger of such an occurrence is great enough to make a known pelvic inflammation in any stage an absolute contraindication to intrauterine radiation in myomata.

Radiation in ovarian growths is worthless, with the possible exception of external radiation in some forms of inoperable ovarian cancer.

Essential Hemorrhage.—Finally we come to that great group of cases which we may term "essential hemorrhages," or hemorrhages without marked gross pathological lesions. With all due respect to the value of radium in the treatment of uterine cancer, it is in the treatment of these hemorrhages of benign origin that radium has won its greatest triumphs in gynecology. Admittedly the classification is bad. From the etiological and pathological standpoints many diverse and dissimilar conditions are, for convenience, grouped together. But from the therapeutic standpoint they all have one feature in common: the symptom of hemorrhage can be relieved, either temporarily or permanently, in practically all of them.

But here again rises the question of whether it is best thus to treat all cases. Manifestly it is not. The question of the age of the patient is here also of paramount importance. The same considerations, stressed in the treatment of myoma, here hold good. Radium radiation in such dosage that the menopause ensues must be regarded as a major procedure, from the standpoint of the patient. When major procedures

come under consideration as a means of checking hemorrhages in girls and younger women supravaginal hysterectomy, with preservation of the ovarian function, is a less drastic treatment than radium radiation and is the procedure of election.

Assured diagnosis, as nearly absolute as it can be made, together with elimination of possible complicating disease of other organs is here also a prerequisite. Of additional importance is the question of malignancy. This should always be cleared up by a preliminary diagnostic curettage before radium is used.

Other Considerations.—Radium has been used in a relatively small number of conditions other than the three great groups just mentioned. It is sometimes advocated as a simple means for inducing sterility. Its use is justified only when operation is contraindicated, on account of the same question of the artificial menopause.

Malignant growths of vulva and vagina, relatively few in number, are best handled by radium except in, possibly, the very earliest cases.

In some clinics chronic cervicitis, urethral caruncle, and pruritus vulvae are apparently satisfactorily treated by radium. Such successes have not been invariably duplicated outside of these clinics, and probably the majority of gynecologists would use other methods of treatment.

Province of the Gynecologist.—Among the various points which have been superficially touched upon, that of greatest importance is, I think, the question of careful gynecological examination and accurate diagnosis. The technic of radium administration is usually easy; ridiculously easy when compared with the technic of an extensive complete abdominal hysterectomy. But from the patient's standpoint it is, barring the operative risk, just as important as the surgical procedure.

Properly to carry out such treatments involves more than the possession of the requisite amount of radium and the surgical tools necessary for its introduction. It demands, first, a thorough knowledge of radium, its physical properties, its possibilities for good and evil, and the technic of its administration. In addition, it demands the same knowledge of gynecological pathology, the same skill in examination, and the same balanced surgical judgment that is needed for the surgical treatment of gynecological conditions.

This by no means excludes the radiologist, the general surgeon or the general practitioner from using radium in gynecology. But it does mean that the radiologist, surgeon or practitioner who takes up this work should also possess the same skill, knowledge and judgment in this field that we naturally expect to find in the trained gynecologist.

Radium treatments can, of course, be given by the cooperation of gynecologist with radiologist. An ideal team of two such men, accustomed by past experiences and mutual understanding to work efficiently together, could naturally produce as good results as could be obtained by any other method. In the long run, however, best results will be obtained when the clinician himself masters the radium technic and personally carries out its administration.

Gynecology has no more place for radium therapy as a distinct specialty than it has a place for specialists in the use of pessaries or uterine dilators. Radium is merely one of the many therapeutic agents at the command of the gynecologist, to be used or not as his judgment dictates.

DISCUSSION (*Abstract*)

Dr. Lawrence R. Wharton, Baltimore, Md.—I shall limit my remarks to cases of fibroids. For two reasons it seems to me that surgery in the large majority of cases is preferable to radium in the treatment of fibroid tumors. I think Dr. Holden suggested this in his paper.

In the first place, surgery offers the opportunity to be conservative. And that is commendable. It seems to me that one should regret as much the necessity of taking out the ovaries in a young woman by surgery as he would their destruction by radium. Surgery gives one the opportunity to save these organs and yet be thorough in treatment. It also gives the opportunity to use myomectomy occasionally, which is another conservative procedure. Also, in perhaps 25 to 45 per cent of cases of myomectomy, there will be pregnancies afterwards. For that reason, in the first place, I prefer surgery in cases of myoma.

In the second place, the pathology is complicated.

It happens that just this fall I completed an analysis of the pathological findings in 500 consecutive myoma cases at the Johns Hopkins Hospital. In this series are both private and public patients, and all the material was studied with meticulous care in the laboratory of Gynecological Pathology. In summary, in only 14.4 per cent was the myoma uncomplicated, and in some of these simple cases the tumor was so large that it reached the xyphoid process. In this series of myomata uteri, most of the complications are surgical problems. Pyosalpinx, salpingitis, tuberculosis, dermoid cysts of the ovary, chocolate cysts, endometriosis, papillary cysts, outspoken early cancer of the endometrium or ovary, tubal pregnancy, calcification and various degenerative processes in the myoma, are some of the complications that you also will find in 500 myomas. We might also mention the numerous cases of appendicitis and gallbladder trouble that can be detected and cured while the pelvic operation is being performed.

Furthermore, it is extremely difficult to discover these adnexal, intrauterine or appendiceal complications be-

fore one opens the abdomen, because the large myoma itself makes accurate palpation and differentiation impossible. If one could be certain of his diagnosis in cases of myomata, then one could use radium with much greater mental assurance. This, however, is not the case, for there is hardly any group of gynecological cases that are as full of continual surprise as the old fashioned myoma.

Because, therefore, surgery offers one the possibility of conserving pelvic organs, of recognizing pathological processes and treating them with thoroughness, I believe that the routine treatment of myomata uteri should be surgical. In making this statement, however, I do not wish you to get the impression that I never find any use for radium in these cases. On the contrary, without going into too great detail, there are several groups of cases in which the employment of surgery would be just as radical, risky and unnecessary as the use of radium is conservative, safe and gratifying. In these interesting cases, it is the function of the trained gynecologist to make the correct and unbiased decision.

GASTRIC CARCINOMA WITH ESOPHAGEAL OBSTRUCTION*

By C. LARIMORE PERRY, M.D.,
Miami, Fla.

An instructive case of carcinoma of the stomach recently came under my observation, and because of the symptoms, together with the operative findings, I feel that the case merits detailed description.

On December 4, 1929, a married man 38 years old came to me complaining of epigastric pain and dysphagia. His family history and personal history were negative up to the onset of the present illness, six months prior to his consulting me.

The gastric symptoms began in July, 1929. Employed as a fireman, he attributed the dull gastric pain, situated under the xyphoid process, to over-exertion in lifting ladders and other fire-extinguishing apparatus. At its onset the pain was not severe, but a sudden dull ache that came on with regularity a few moments after meals. The ache would disappear in from two to four hours after food intake. This same gastric discomfort persisted for two months, when the character of the pain changed, becoming more severe. As soon as the patient ate a few mouthsful of food he experienced a sharp pain in the epigastrium, followed by nausea. He soon learned that vomiting gave immediate relief of pain, and observed that he was relatively free from all gastric discomfort when his stomach was empty. During the latter part of November the patient noticed streaks of blood in the vomitus, and also that his stools were black. Liquid foods gave him considerable trouble, as they seemed to "hang in his throat," and it was with difficulty that he could retain them.

One month prior to his consulting me, the patient had a very severe attack of pain lasting several days and confining him to his home. The whole upper abdomen was very tender, and the pain was referred into the back and to the right scapular region. He was unable to eat because of the severity of the reaction following food intake. Hot compresses and linaments were applied for relief.

Aside from the gastro-intestinal history given above, there was a marked loss of appetite. The patient felt that he could eat, but knowing that food would immediately cause pain, he resorted to the only protective means he had of keeping himself comfortable. He employed high powered cathartics to correct an obstinate and progressive constipation.

Physical examination revealed an anemic, undernourished man weighing 112 pounds, against a normal weight of 155 pounds six months previously. The systolic blood pressure was 118, diastolic 82, pulse 76, and temperature normal. The head, neck and chest were negative, with the exception of exaggerated breath sounds in the left chest, and a few mucous rales. The teeth were in good condition. The abdomen was scaphoid, and only on firm epigastric pressure was pain elicited in the region of the xyphoid. No masses were palpable. The genitalia, spine, joints and extremities were negative.

Laboratory findings showed a negative Kahn; and urine with a specific gravity of 1.031, containing a few granular and hyaline casts. The blood examination showed: hemoglobin 71 per cent, red blood cells 4,410,000, leucocytes 11,400, neutrophils 57 per cent, lymphocytes 38.5 per cent, eosinophils 1 per cent, basophils 1.5 per cent, monocytes 0.5 per cent. There was a slight anisocytosis and poikilocytosis. The test meal was significant: total acidity 24, no free hydrochloric acid, no Boas-Oppler bacilli nor lactic acid, small food remnants, fresh blood II, mucus III, and occult blood III on a basis of IV. X-ray following a barium meal revealed a delay of the media at the cardia, with moderate dilatation of the esophagus in its lower one-third. A filling defect in the cardia on the lesser curvature suggested ulceration or new growth. The chest x-ray was negative.

A preoperative diagnosis of perforating gastric ulcer or carcinoma with mechanical esophageal obstruction was made, and the patient was hospitalized. For five days he was fed on a modified Sippy diet, with the fluid intake forced up to between three and four liters daily. A silk thread was swallowed on the day preceding the operation, and left in place during the surgical procedure.

Exploration was done under local anesthesia and nitrous oxide, through a left rectus incision. The liver was sharp edged and grossly normal. The gall bladder and appendix were negative to gross inspection and palpation. The stomach was high lying, being in its entirety above the left costal margin. From the angle of the stomach to the duodenum the organ was as normal as could be hoped for. On palpating the lesser curvature, the hand came in contact with a hard, irregular mass the size of a hen's egg. When followed around toward the greater curvature, the mass was found completely to encircle the esophageal entrance into the stomach. Aside from the cavity in the center of the growth, which I attributed to be the opening of the esophagus into the stomach, no definite crater could be felt. An enlarged gland, removed from the gastro-hepatic omentum for diagnosis, was reported adenocarcinoma. Had the condition been reported inflammatory, the treatment would of necessity have been

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the same, due to the inaccessibility of the growth to surgical intervention, and its slow encroachment into the lumen of the esophagus. The wound was closed in the usual manner.

Convalescence after the exploration was rapid, and for some reason the dysphagia disappeared. On the fourth day the patient was given a high caloric soft diet, which he retained with no pain. He was allowed to go home on the eighth day, and on the fourteenth postoperative day was up and about the house.

A microscopic section of the gland removed was sent to Dr. Broders of the Mayo Clinic for grading of the malignancy. His diagnosis was a grade three adenocarcinoma with a life expectancy of three to four months.

The case is instructive only because of its involvement of the esophagus, and its clear cut history of dysphagia which closely simulated cardiospasm.

WHOLE TIME MEDICINE*

By Roy K. Flannagan, M.D.,†
Richmond, Va.

Medical Standards.—The art of medicine is the exponent of truth in philosophy and in science; and if it is honorably practised in accordance with ancient and accepted standards it exemplifies altruism and social service in their highest form. Though, of necessity, containing many ramifications, divisions, and specialties, medicine is essentially one.

It is my purpose in this paper to set forth as clearly as I can, the logic of the situation of the practitioner of medicine in a changing time, and to show the obligation resting upon him to practise whole medicine according to the ancient formula, whatever be the branch of it with which he is best qualified to deal, and to fix beyond cavil his status as a preventive agent first of all.

Preventive medicine, if it is a specialty of our noble art, and it has been so considered for a long time, is so because under the stimulus of the altruistic impulse and tradition of their calling medical groups have been largely instrumental in thrusting certain of their number, whom they have deemed qualified, out into state and community health service, charged with organizing both the profession and the public to abate the source of the disease calamities that cry insistently to the profession for cure.

The Scope of Medicine.—The art of medicine has its roots buried deep in antiquity and the most ancient recorded knowledge of it bears clear evidence of high appreciation of public duty and private obligation.

The great Hippocrates, the author of the oldest medical treatise extant, the father of medicine, whom all regular physicians hold in peculiar honor and reverence in specifying the duties of the medical man, placed the preservation of health ahead of the practice of the healing art.[1]

Lord Francis Bacon, whom Alexander Pope, in his essay on man, called the wisest of mankind; who was thoroughly conversant with ancient medical lore, and whose treatises on medical subjects, particularly medical philosophy and ethics, have served as a standard for centuries, said in elaboration of the doctrine of Hippocrates: that medicine consisted of three parts, the preservation of health, the cure of disease, and the prolongation of life.[2] This definition has never been challenged, and yet it definitely places preventive measures well in advance of curative among the duties of the physician. To separate these duties into specialties and to give a less dignified place to preventive medicine is not only to be historically incorrect, but illogically to confuse social values.

Medical Arrogance.—That there is much confusion of thought in that regard is stressed by Dr. Louis I. Harris, of New York, in his Chairman's Address before the Public Health Section of the American Medical Association at Minneapolis last year (1928).

He remarked upon "The haughty aloofness of certain physicians from interest in participation in health work," and "a certain air of condescension, if not utter disdain, for public health work" on the part of others. He cites this "to illustrate the excessive lengths to which specialism has gone, or else as evidence that the physician who disclaims interest in public health is merely a high grade mechanic without the quality of mind and heart to entitle him to take the name physician."

The experience of Dr. Harris is that of many health officers and they cannot but feel that such arrogance is peculiarly out of place, not only on the grounds noted by Dr. Harris, but on the basis of actual self-preservation. "Shall the eye say to the hand, I have no need of thee?" It is quite obvious that any attempt to dissociate the prevention of disease from the general medical profession would be much more disastrous to the specialist in cure than to the public health specialist. Public health departments are required by the public, on the basis of facts furnished by the medical profession and by medical research, to stop communicable disease in its tracks whenever it appears, to abate the conditions which make it possible and to promote to

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*Read in Section on Public Health, Southern Medical Association, Twenty-Third Annual Meeting, Miami, Florida, November 19-22, 1929.

†Assistant State Health Commissioner.

the fullest extent abundant health and length of life, a duty which all will recognize as a paramount public service. That anyone should hold a medical public health officer in less repute because he specializes in health and longevity, rather than in cure is diverting; but for a physician whose income is dependent to a great extent upon the existence of human physical distress to be disdainful of a physician employed by the state or locality to prevent that distress is absurd.

Medical Perspective.—While it is clear to everyone that the general principles involved in the work of disease prevention are medical principles and that cure is but an incident in the application of most of them; the physician's time and attention in the past have been so engaged by demands upon his skill in curing that his perspective in many instances has been lost or badly dislocated. Now these demands have lessened so that he frequently has more time to look about than he needs, and lack of proper focusing ability is causing the reactions which are to be expected from an astigmatic, but are nevertheless as futile as trying conclusions with a steam roller.

In the evolution that is taking place in our civilization consequent upon the increasing ease of communication and the wider dissemination of knowledge, the same phenomenon is apparent that geologists and anthropologists tell us has taken place in the evolutionary process throughout time, in which forms of life that could not adapt themselves to a changing environment disappeared and gave place to other forms that could. Businesses, institutions and occupations under like pressure must yield to the same law. Those who cannot, or will not, adjust their thinking of their practice to the demands of a changing time will inevitably go down in the struggle. The practising physician will thoughtfully consider the whole situation and govern himself accordingly. It is manifestly impossible to check a glacier, stop the tide or turn back the clock of time, and it is unintelligent to try. Fortunately, evolution is slow and there is ample time for an adjustment that completely accords not only with all that is best in medical thought and tradition but is equally in line with the highest standards of public service and private right.

The Health Department.—Whenever in a democracy it is borne in upon the public mind that a certain practice or procedure is of such universal worth as to be a public necessity, which yet, under existing circumstances, is not economically available to all the people; the state following public demand takes up that procedure and applies it in the public interest, sometimes well, sometimes ill, as is the case in all human affairs.

Thus it is that measures for the prevention of disease and the promotion of the public health, having graduated, so to speak, from the school of the research worker and the doctrinaire, have entered civic life and gained on merit a definite place in practical politics. In other words, the state having gone into the health business through the establishment of health departments, has by that action proclaimed that disease is no longer to be considered a private or local matter, but one of grave public concern, and the physician who has hitherto dealt with disease as a local matter, that is, strictly between himself and his patient, must for the public good bow to the public will, and as the one best qualified to aid, find his proper place in all plans for completely conquering his ancient foe.

The department of government for fighting disease and promoting health is too new in the general scheme of things as yet to have adjusted itself completely, or even to have availed itself of all of the forces that should be coordinated with it. At first, because of his status as initiator and pioneer, the physician's interest and aid was taken for granted. Then, as time went on, and this as a general proposition was found to be an unwarranted assumption, the health officer on the one hand and a considerable proportion of general practitioners on the other, became critical, more or less dogmatic, and independent in their attitude toward each other, with a resultant loss of sympathy for each other's problems and points of view.

Mutual Cooperation.—Fortunately, thoughtful men in all divisions of medical science see this drift and may be counted on to aid in checking it. Fortunately, too, health department practice is not yet old enough to be hidebound, nor are its leaders unmindful of the debt that preventive medicine owes to the general practitioner, nor of the paramount service he renders in promoting its success. The general practitioner, however, and those who presume to speak for him, should fully realize that no amount of "boneheadedness" on the part of health administrators here and there or any lack of cooperation on the part of any physician, or group of physicians, will for an instant delay the progress of the gen-

eral movement for the abolition of communicable disease.

Relative Values.—Cure is relatively unimportant from the standpoint of the public, for the cure of an individual by a physician is of little consequence in comparison with the rapid disappearance of a deadly disease in the community by the application of preventive principles.

This is made glaringly apparent now that state and municipal authorities and more important still, chambers of commerce, are becoming aware that public health protection is considered a necessity by the persons to whom their appeal is specially directed, and, therefore, it must be developed to complete adequacy irrespective of lesser considerations. The public is awake. The great American Goddess, Prosperity, is on the throne. If the physician remains asleep, he is likely not only to lose her smile but his own prestige as well.

Does acceptance of the situation by the physician with calmness and good sense involve loss of dignity or caste? I think not.

The Honor Roll.—The outstanding personalities of the medical and scientific world have rarely been exponents of cure alone, but were and are in the main, those who have sought to restrict in wholesale ways the need of cure. Such were Jenner, Pasteur, Lister, Virchow, Koch, Eberth, Osler, Ross, Reed, Carter, Gorgas, and a host of others, living and dead. There is no physician who does not wish in his innermost soul to reach by similar means such heights of public and professional esteem.

Why should he not make the effort? It is, I believe, entirely possible for the medical profession to find a profitable, dignified place in the scheme of disease prevention, as well as to continue to meet the demands of a civilization that will never be sufficiently well trained or organized to do without the healing touch.

"In Union, Strength."—A close interrelation of the work of the general practitioner with local health work would be of great advantage to the public, and if it be definitely desired by organized medicine, no insuperable difficulties stand in its way. Programs of preventive medicine could be prepared involving the assignment of certain activities to practising physicians in given areas, on acceptable terms.

With physicians better trained year by year in public health principles and practice, and with organized medicine participating constructively in public health programs and activities, the public will much more readily be brought to pay what the service is worth.

The point I seek to drive home is that improvement of the physician's situation lies, not in unenlightened antagonism toward public health tendencies, nor in the repudiation of ancient principles that have kept the honor of the profession high, but in a finer quality of public service where the physician's health department contacts will no longer be considered irksome or unimportant by him, but as a duty involving the very essentials of a healthy and completely functioning social organism.

The public, though generally surprisingly alert where its interest is concerned, is yet very easily misled in medical matters and succumbs readily to the wiles of the charlatan. It is imperative, therefore, that the branch of medical science most in touch with the public shall not be isolated in any degree from the rest of the profession.

From what has been said it ought to be fairly clear that the public has "the bit in its teeth," so to speak, and nothing but the most rational handling of the situation will save even a measure of control of the reins to the physician. He ought to be best qualified to guide in such a crisis. Shall he not demonstrate his fitness?

Historical Perspective.—The physician belongs to what has been long classified as a learned profession. In my judgment he has but to prove the truth of that classification in order to keep command of the situation. If he will hark back to his literary and philosophic beginnings and practise medicine wholly, according to the Baconian formula instead of in part as at present, he will, I believe, not only remain in the saddle, but will incidentally save both his prestige and his pocketbook. He is blind, indeed, if he does not see that one or the other, perhaps both, are in grave danger.

If the physician's research into medical lore leads him into favorable consideration of what is invidiously called state medicine, let him not be alarmed. He has not necessarily fallen from grace. From whence, may I ask, does the edict against state medicine come? Who wrote it and from what source does it gain its authority? I can find no pronouncement against it nor anything like it in the writings of the fathers of medicine, nor in any medical author of note in modern times. In fact it would seem that the best medical thought and tradition is all for making public and popular every means rendering mankind healthier and longer lived, and for

frowning upon any scheme that would patent or circumscribe medical knowledge.

State Medicine.—If the state can devise a method for more completely popularizing medical truths and making health universal, and the need for cure negligible, and by taxation and organization, and the employment of skilled physicians make such a plan effective, what physician can raise objection without stultifying himself and abandoning every principle upon which medical ethics and honor rests? Understand me, I am dealing with a purely hypothetical question. If plans are suggested with such an object in view, can the physician in honor withhold his sympathy and aid?

Medical Honor.—The medical profession has long enjoyed the character of a noble, disinterested, self-sacrificing institution for the reduction of human woe, through the prevention and cure of physical ills with which man is afflicted.

On that character it has successfully appealed for public support of medical education; shall the catch-word "state medicine," be permitted to obscure and perhaps destroy this the greatest glory of the profession? If the idea became general among physicians that they must strenuously resist every effort on the part of the public to bring competent medical service within the reach of everybody (an utterly impossible contingency in my opinion), it would constitute a grave social menace that would leave the physician impotent and discredited in a world that needed his enlightened guidance more than ever.

The exploitation of human misery as a means of livelihood is inherently an unsocial and disappearing concept; a concept that in so far as it applies to the physician, would long ago have brought him into general disrepute but for the exalted standards by which his conduct has been guided. When those high standards go the independent physician goes with them.

CONCLUSIONS

Let us then, as modern exponents of the art of medicine as defined by Hippocrates, the philosophy of medicine as outlined by Sir Francis Bacon, the science of medicine as set forth by Sir William Osler and the practice of medicine as exemplified by Edward Jenner and hosts of public spirited physicians, who, through the centuries have devoted their lives to the well being of their patients not only, but to the larger good of the communities, states and nations that gave them birth; resolve from the depths of our souls to practise whole time medicine according to the ancient formula. Let us, with all the emphasis of which we are capable, say:

"I will lend no part of my influence to any plan or proposal that tends to obstruct the progress of the most fundamental and important part of my art, namely, that devoted to the prevention of disease and the promotion of health and long life among men, but, as a lover of my fellow man, as a doctor, a citizen and a patriot I am prepared to become even the servant of the state if, thereby, the purpose for which every true physician exists can be the earlier achieved."

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DISCUSSION (*Abstract*)

Dr. J. A. Ferrell, New York, N. Y.—The subject introduced by Dr. Flannagan has been before the medical and health conventions for several years. The movement for the employment of full-time health personnel has gained considerable headway. It was supported in the beginning by the medical profession and continues to have this support. Now that between 200 and 300 cities, and between 400 and 500 counties have full-time medical health officers, although the development is still young, medical men are beginning to wonder just how far the conduct of service in preventive medicine will extend. As the practitioners encounter a steadily diminishing number of cases of communicable diseases, they are naturally concerned as to the part they can play in the field of prevention. Fortunately, the proponents of public health service are advocating more and not less service by the physicians as well as by the health workers. The objective is to get the work done as far as practicable through a direct relationship between patient or family and physician and to avoid unnecessarily increasing the volume of work to be done at public expense.

The medical schools of the past generally have not given thorough courses, as Dr. Leathers points out, to the medical students with regard to preventive medicine. Increasing interest, however, by physicians already in practice, and by certain of the medical schools in training the students for the practice of preventive medicine, is being manifested. Assuming that the physicians are qualified or will be qualified to practise preventive medicine, to examine and advise the apparently healthy with a view to keeping them in health, the problem that will have to be worked out which is now a source of deep concern to physicians is how and to what extent they are to be compensated under the new order of things. Their investment of time and money in preparing themselves to practise medicine has been very heavy and consequently if they render a high grade of service they are entitled to receive a decent income. As the health workers and the physicians have a common cause and, as I see it, no really conflicting interests, it would be the part of wisdom for them to work together in evolving a plan which organized society will consider satisfactory and reasonable. Failure to do this may be distinctly injurious to the cause of public health, to the medical profession and to the health workers.

Dr. A. T. McCormack, Louisville, Ky.—Recently I had the privilege of hearing statements before the For-

eign Commerce Committee in the House of Representatives in a hearing on Maternal and Infant Welfare. I was interested when representatives of our profession, who are specialists in obstetrics, stated that the general practitioners graduating in medicine are wholly untrained in obstetrics and that there were less than 250 competent obstetricians in the United States today.

We are all in danger of getting in just that attitude regarding our own special branches of medicine. The congressmen and other persons attending the hearing were amazed. Their whole faith in medicine as a profession was shaken.

The same thing is true if we become too critical of other physicians in the general practice of medicine who do not know as much about our specialty as we do. Incidentally, that is the reason we are specialists. It is our job and responsibility to see that the general practitioners know enough about our specialty to recognize and practise preventive medicine. We are the ones who are expected to lead the teaching in that respect.

Dr. Flanagan (closing)—Since writing this paper I have seen an article in the *Chicago News* and an editorial in the *Saturday Evening Post*, both bearing out the truth of the statement that there is a growing unrest. The medical profession cannot afford to be divided in policies if it is to meet the demands of a progressive age.

DR. J. MARION SIMS*

By CLARENCE K. WEIL, M.D.,†
Montgomery, Ala.

Robert Louis Stevenson once remarked that there are certain groups of men who stand out above the masses: the soldier, the sailor and the shepherd not infrequently; the artist rarely; the physician almost as a rule. And yet among this group of men whose daily life is devoted to the relief of suffering, there are few whose names will go down in history; for it is a peculiarity of mankind that he does homage and gives praise more often to those who have destroyed life than to those who have conserved it.

On the lips of every one are the names of great generals and conquerors, yet few who are not connected with the medical profession can name five physicians, who by their imagination, their persistent investigation and their foresightedness, have made our Country a safe and healthful place to live in. Gone are the dreads of malaria and yellow fever, of cholera and hydrophobia. Rapidly disappearing are the ravages of typhoid and tuberculosis. Diphtheria and tetanus occur far less frequently than in the past. No longer are diabetes and pernicious anemia incurable diseases. Each of these steps in the advancement of medical science is linked with the name of some man who is entitled to immortal fame. If we, of Montgomery, can claim as our own one who has contributed his share toward the alleviation of human suffering, then we have just cause for being proud. Such a man we can claim as our own, and it is to do homage to him that we are gathered here today.

Eighty-five years ago, there stood on the spot now occupied by these buildings the office and infirmary of Dr. J. Marion Sims. He was born in Lancaster County, South Carolina, January 25, 1813, the son of John Sims and Mahala Mackey. After receiving his primary education in the local schools, he went to Columbia University, in South Carolina, and graduated in 1832. He began his medical education by reading under the direction of a local physician, attended lectures at Charleston, and subsequently studied medicine at the Jefferson Medical College in Philadelphia. After a rather short and unsuccessful attempt at practice in his own town, he came to Alabama and settled in Mount Meigs. During the Creek war, he served in the capacity of assistant surgeon. Two years later he went to Cubahatchee Creek, but being stricken with a severe and persistent case of malaria, he left the country and took up practice in Montgomery. Up to this time his career had not been remarkable, but it was in our city and on this very spot that he made the discovery which entitles him to fame.

Women, at child-birth, often suffer from wounds as characteristic as those received by soldiers in battle. Certain of these leave the unfortunate woman in a state so miserable and loathsome that she becomes a burden both to herself and to her loved ones. Many of these women have prayed in vain for death to relieve them of their suffering. The man who could discover a method of curing these women would be, indeed, a benefactor of the human race. Such a man was J. Marion Sims.

In the year 1845 he had been consulted within a period of a few days by three of his friends who had slaves suffering from this condition. He realized that their cases were hopeless and would have declined to treat them had he not accidentally discovered

THIS TABLET MARKS THE SITE OF
THE OFFICE AND INFIRMARY OF
DR. J. MARION SIMS
HERE, IN 1845, HE PERFORMED THE FIRST
CLOSURE OF A VESICO-VAGINAL FISTULA WITH
WIRE SUTURE, USING A PEWTER SPOON AS
SPECULUM. THIS OPERATION MADE HIM
FAMOUS THROUGHOUT THE WORLD.
PLACED BY THE
MONTGOMERY COUNTY MEDICAL SOCIETY
UNDER THE AUSPICES OF
THE ALABAMA ANTHROPOLOGICAL SOCIETY
A.D. 1930.

Bronze tablet at 21 South Perry Street, Montgomery, Alabama, unveiled April 16, 1930. The tablet reads: "This tablet marks the site of the office and infirmary of Dr. J. Marion Sims. Here, in 1845, he performed the first closure of a vesico-vaginal fistula with wire suture, using a pewter spoon as a speculum. This operation made him famous throughout the world. Placed by the Montgomery County Medical Society under the auspices of the Alabama Anthropological Society, A.D., 1930."

*An address delivered April 16, 1930, on the occasion of the unveiling of a bronze tablet erected by the Montgomery County (Alabama) Medical Society, marking the site of the offices and infirmary of Dr. J. Marion Sims.

†Immediate Ex-President, Montgomery County Medical Society, under whose administration tablet was arranged for.

that by means of a bent pewter spoon he could inspect these wounds. Sims was thus the first man who saw one of these wounds and the first sight of them convinced him that they could be cured by operation. Into his little hospital he gathered half a dozen negro women who were suffering from this condition, and there he put into practice the operation which he had devised. His first operation was a failure, as were his second and third, but undaunted, he persisted over a period of four years, and after performing forty operations on one of his patients, he was rewarded by success. In spite of the ordeal through which she went, I feel certain that this poor old negro woman would have gone through twice as much in order to become cured of her condition and I doubt that any man has ever been more joyful than was Dr. Sims when his efforts were, at last, crowned with success. Using a pewter spoon as speculum and a silver wire as suture, he had succeeded in curing, for the first time in medical history, a vesico-vaginal fistula.

The rest of his life is a story of physical suffering and a struggle against jealousies and antagonisms to perpetuate his ideas. Compelled by his ill health to move to a cooler climate, he took up his residence in New York City and after severe trials and great humiliations, he finally succeeded in establishing a hospital in which he could perform his operation for the relief of suffering womankind. That hospital was the Woman's Hospital of the State of New York.

In 1877 Dr. Sims returned to Montgomery to visit his old friends and the scene of his discovery. What a contrast between his first arrival in Montgomery and this triumphant return. He describes his first arrival in these words, "I was young then, inexperienced, in bad health, and very poor. I had nothing whatever to recommend me, nothing but honesty, industry, and a determination to succeed. Your Crommelins and Pollards gave me houses to live in until I was able to procure one for myself. Your merchants gave me credit for food and raiment for my family, when I had not a dollar to pay for them." On this occasion a banquet was held in honor of Dr. Sims by the Montgomery County Medical Society, on whose roll his name had appeared. In the address on that occasion, Dr. W. O. Baldwin described his illustrious confrere in these words: "He has risen, by the splendor of his own genius, above all obstacles; and before he has reached the meridian of life, we find him one of the acknowledged discoverers and benefactors of the world, and ranking as one of the foremost men in this Country. In the great capitals of Europe he has been the guest and pet of emperors, he has received honors and distinctions from learned scientific bodies, he has been courted by the elite in his own profession, sought by the nobility, and has received titles and decorations from courts of the largest empires in Europe." What a contrast! What a journey from the days when slaves and free negroes were his patients to those when the nobility sought his advice.

I feel that no one can learn more from the story of Dr. Sims than can those men who have but recently entered upon the practice of medicine. He has taught us that location is no bar to the attainment of fame and success. The Montgomery in which he worked was far smaller than it is now and the handicaps under which he labored were far greater than those which we must overcome. If in the early days of practice, our clients come from the poorer walks of life, we cannot help but be encouraged by the thought that he who was sought by the greatest in this country and in Europe made his start among slaves and free negroes and that it was with these poor people that he worked out his speculum and his operation which made him sought after by the nobility and which has given him everlasting fame.

I know of no group of men who should take greater pride in the erection of this tablet than the members of the Montgomery County Medical Society, of which Sims was once a member and which has shown its interest by sponsoring the erection of this tablet. And I know of no citizen of this beloved city who can fail to be proud that one whose name has become immortal lived and worked and attained his fame here in our midst.

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EDITORIAL DEPARTMENT

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TECHNIC OF CHOLECYSTOGRAPHY

Since the introduction of the Graham test for gall-bladder function, there has been a misconception on the part of not a few physicians in that they have considered failure of the gall-bladder to be visualized as a pathognomonic sign of disease of this organ. This is contrary to any claim made by its originator. By following the statements made by Dr. Graham one can see that it is a functional test. This makes it all the more necessary to use a standardized and carefully controlled process in applying this diagnostic method.

The conflicting reports of its value in the presence of pathological change may be evidences of a difference in the manner in which the test is carried out by radiologists, and not entirely a reflection on the method itself. Varying reports are submitted as to the advantages of the oral as compared with the intravenous method of giving the dye; some of these condemning the latter method because of the severe reactions which occur in a large percentage of cases. The recent article in the *American Journal of Roentgenology and Radium Therapy*, January, 1930, by Drs. C. A. Waters and J. H. King, showed that in 96 cases, no serious discomfort to the patient was encountered in the intravenous method; the contraindications in this series being limited to cardiac decompensation and uremia. In this paper they give in detail their method of intravenous injection. Case, of Chicago, and Moore, of St. Louis, use the same method.

The intravenous method is, of course, the one of choice in investigative work if it can be carried out with the proper technic; but it is believed by a great many that the oral method will remain the one of universal application by the average physician who cannot hospitalize his patients while studying them; and for that reason, one should strive to perfect his methods of application of this procedure.

Fellows, of Cornell Clinic, has found that preliminary dieting before applying the test induces better and more uniform results. The patient is given a purgative the night before the dye is administered. The next day a diet consisting of the following is instituted: for breakfast, orange juice, cereal if desired, buttered toast, cocoa, or coffee; for lunch, soup, tea, and crackers; for dinner, two soft boiled eggs, buttered toast, milk, ice cream, and plain cake. For convenience the dinner is usually taken at 5:30 P. M. and two hours later the dye is given (phenoltetra-iodophthalein 2.5 grams). The patient is instructed to go to bed and eat nothing until after the first films are made the following morning at 10:30 o'clock. Often the shadow is missed because the films are made too early. The normal gall-bladder shows its greatest concentration 15 to 18 hours after injection of the dye.

The importance of the proper position; making exposures with respiration suspended; and using uniform penetration, can not be overestimated. It is essential to use multiple cholecystograms, for by comparison of these one can at times differentiate extracholecystic deformities such as gas in the duodenum or other parts of the gastro-intestinal tract from changes in the gall-bladder outline itself. A preliminary film before the dye is given should always be made.

When using the oral method, evidence of dysfunction should be checked by a repetition of the test, as not infrequently normal function is demonstrated on the second examination.

After giving the routine meal of egg yolk, it is necessary that radiograms be made with the same careful technic as before. It has been demonstrated that the normal gall-bladder empties approximately three-fourths of its contents the first half hour after this routine meal and

it is entirely empty within one hour. Some authorities claim that films should be made every fifteen minutes until the gall-bladder is empty. The gall-bladder should, by all means, be followed until there is no shadow present.

In few diagnostic procedures is proper technical regulation of more importance than in making the phenoltetra-iodophthalein test. Unless carefully controlled, it may be a liability causing a diagnostic error.

WHITE BREAD AND BROWN

At the end of the Nineteenth Century, when the medical sciences were dominated by bacteriology and the newly discovered technic of aseptic surgery, white-furnished hospitals and operating rooms, white-clad nurses, surgeons and orderlies, white flour, white sugar, and white fats came into popularity. Natural foods were bleached and extracted and done up in sterile packets to conform to a hospital standard of bacteriological purity. Material of a darker hue in food substances suggested contamination and dirt. Foods of this kind readily supported life of micro-organisms, and were frequently spoiled in shipping and storage. The public, including most of the present generation of physicians, was then positively conditioned to white bread stuffs in its infancy; and food habits once formed are difficult to change, in poultry, animals, and men. The public opinion which demanded a clean food supply became an important factor in the decrease of many infectious diseases, and in the reduction of the mortality rate. It had also its disadvantages.

The Twentieth Century has been dominated rather by the concepts of biochemistry than of bacteriology. As the science of nutrition has progressed, the inferiority of white flour, polished rice, and similar food stuffs, their inadequacy to support life, has become apparent, and is freely admitted even by those who advocate consumption of white breads on the ground of expediency. Because the use of highly milled cereals is an established and convenient custom, they continue to be popular among well informed persons, who hope that their manifold deficiencies may be elsewhere attoned. White flour is cheaper than whole grain flours, because it has keeping qualities which whole grains lack. Micro-organisms can not readily live on it. Hence its popularity with food merchants who always have difficulty in finding a ready market for perishable commodities. The educated palate of the average person today is accustomed to it, and the average person prefers white to whole grain flours.

Since white flour products form quite a large part of the average dietary, and since their deficiencies are admitted by all, the best practicable means of supplementing them should be sought.

Morgan and Barry,[1] of Berkeley, California, have incorporated into a standard white luncheon roll supplied to high school children, a large per cent of wheat germ, which they say is rich in vitamin B, the antineuritic vitamin. Fruits, according to these workers, are poor in vitamin B. Whole cereals are the richest source. Groups of malnourished high school children who were observed over a period of several months ate wheat germ rolls instead of white rolls for their school luncheon, with no other change in the dietary. They were compared with other groups which had the ordinary white rolls, and a considerable increase in weight was found in the groups which had two wheat germ rolls a day. The California workers feel that the decreasing amounts of vitamin B in the modern diet are related to anorexia in children, nervousness, constipation, and other gastro-intestinal difficulties. Wheat germ, they report, may be sterilized like white flour to keep indefinitely, and may be conveniently added to white flour as desired, to make bread.

Small improvements in the dietary frequently favorably influence conditions of severe malnutrition in children.

There are a number of breads on the market to which concentrated vitamins have been added. Yeasts and codliver oil, of course, are prescribed with increasing frequency for many ailments. Vitamins are rapidly supplanting the tonics of the past quarter century as popular agents for blood building and as correctives for malnutrition in childhood and in adults. By biochemical tests it is possible to learn which of these added increments actually does improve human nutrition. White bread is deficient in vitamins, minerals, and many other elements. Wheat germ is apparently a wholesome addition.

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THE SUPRARENALS IN DIABETES

Internists have observed the failure of certain cases of diabetes to respond to insulin and the relatively large quantities of the drug which must be used for other patients. It is also well known that the fall of blood sugar of a diabetic after a definite quantity of insulin is not mathematically predictable. Though lack of insulin seems to be the chief factor in the glycosuria and hyperglycemia of diabetes, and symptoms of the disease respond readily to insulin therapy, many other reactions are involved in the metabolism of carbohydrates.

In the normal course of events, presumably, as insulin increases the rate of combustion of glucose, suprarenalin stimulates the release of glucose from the liver, where it is stored as glycogen. Thus suprarenalin on injection brings about an elevation of blood sugar and prompt relief of symptoms of insulin overdosage. The suprarenal glands apparently act in preventing insulin shock, since animals from which one suprarenal has been removed are sensitized to insulin. In them comparatively small doses of insulin rapidly cause severe convulsions.[1] The suprarenals have a buffer effect, which tends to prevent violent insulin reaction.

Suprarenalin, which is obtained from the medulla of the suprarenal glands, was one of the earliest of the internal secretions to be studied. Among its effects are dilatation of the pupils, acceleration and increased force of the heart beat, constriction of the arterioles and contraction of the sphincters, conversion of the hepatic glycogen into glucose, and stimulation of the sympathetic nerves. These are, as Cannon pointed out, the same phenomena which are observed in emotional stress. Secretion of suprarenalin is increased in states of emotional strain, for example, by a cat at the barking of a dog, after physical exertion, exposure to cold, asphyxia, and during hypoglycemia.[2] It has distinctly advantageous effects in the defensive reaction. Its oversecretion, however, becomes pathological.

Although suprarenalin stimulates the mobilization of glucose into the blood stream, it hinders the next step in sugar metabolism; it assists in the wasting of glucose by causing glycosuria. According to Colwell and Bright,[3] of Harvard, intravenous glucose which is injected continuously into a normal animal is utilized or stored; but if suprarenalin in physiological quantities is given at the same time, all the injected glucose is excreted in the urine. The administration of suprarenalin completely suppresses glucose combustion, even in the presence of an excessive supply of glucose. It brings sugar into the blood under normal circumstances, but when its secretion is abnormally increased, burning of glucose in the tissues is prevented and unchanged glucose is excreted. The condition then resembles that found in total diabetes.

The frequent onset of diabetes or transient hyperglycemia and glycosuria during an emotional strain might thus be due to prolonged secretion of suprarenalin. Some students have suggested that diabetes mellitus, instead of being primarily a pancreatic disease, is caused by the activity of the suprarenals. Contemporary evidence, according to Colwell and Bright, favors the view that ordinary diabetes is the result of a functional disorder of the pancreas which is dependent upon disease of the sympathetic system, and that continuous excessive secretion of suprarenalin may be an important factor in this mechanism.

1. Cannon, W. B.; McIver, M. A.; and Bliss, S. W.: Sympathetic and Adrenal Mechanism for Mobilizing Sugar in Hypoglycemia. Amer. Jour. Physiol., 69:46, 1924.

2. Wright, Samson: Applied Physiology. New York: Oxford University Press, 1929.

3. Colwell, A. R.: and Bright, E. M.: Use of Constant Glucose Injections for Study of Induced Variations in Carbohydrate Metabolism. Amer. Jour. Physiol., 92: 555, April, 1930.

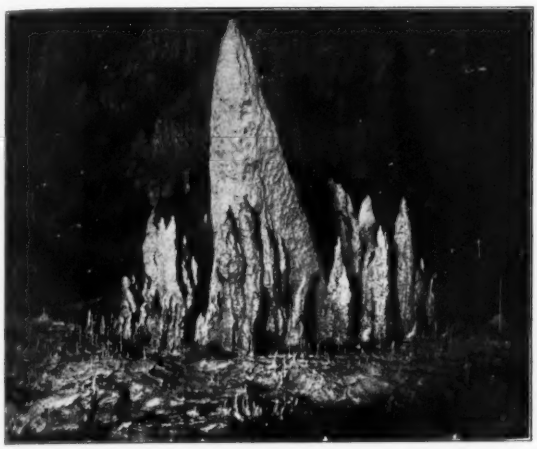
Louisville—Where We Meet

MAMMOTH CAVE*

The holding of the Southern Medical Association's convention in Louisville this year presents an opportunity for attending the convention and at the same time visiting a number of places of international fame. Mammoth Cave, one of the seven modern wonders of the world, the greatest subterranean spectacle known to man, is situated within one hundred miles of the convention city, and being south of Louisville may be visited by those planning to attend the convention with very little additional traveling.

Chance, that harbinger of the unusual, is responsible for the discovery of Mammoth Cave, so the story goes. Back in 1809, a hunter pursuing a wounded bear to its lair stumbled into the cave and thus rediscovered for the white man the cavern which had been known to the Indians, and probably to the prehistoric races before the red man. The hunter, Houchins by name, lost no time in acquainting others with his discovery and ever since that time explorations of its cavernous depths have been going on.

*Prepared by the Louisville Convention and Publicity League, Harry G. Evans, Secretary and Managing Director.



One of the fantastic mineral formations seen within Mammoth Cave.

The need of saltpetre for use in gunpowder in the War of 1812 was responsible for the first explorations of any consequence. The pipes, made of wood, the immense vats and other paraphernalia used in mining the soft soil rich in nitre are still to be seen just as they were left over a century ago.

Down into the bowels of the earth, through the very entrance found by Houchins, the visitor enters into a world different and absolutely removed from every day commonplaces. He is always in the presence of evidence of the mighty power of water; for water it was that untold centuries ago carved out of the limestone these great avenues, cathedral like spaces, grottoes, fantastic rock formations, and other sights which thrill the explorer.

Probably half a mile within the entrance, there are a number of crude stone huts, the remains of an experiment by a colony of unfortunates seeking a cure for tuberculosis. These houses were inhabited a century ago by afflicted persons deluded into the belief that the constant temperature of the cave would cure their malady. Of course the complete absence of sunshine brought an inevitable result, but the tragic remains of a blasted hope are still to be seen in the cave.

Almost four hundred feet below the entrance level the visitor beholds a marvel of nature, an underground stream known as Echo River on which the guides take the traveler for a boat ride of over half a mile. In the waters of Echo River scientists have found eyeless fish, crustaceans, and leeches, their power of sight made useless in the everlasting darkness with the result that nature is dispensing with the superfluous organs. Blind insects, beetles, crickets, mites and gnats in other portions of the cave hold much of interest to the naturalist.

Echo River receives its name from the reverberating sound effects obtained on its surface. The voyager on the river passes under a low arch for a short space and then the roof rises rapidly away from the water and he enters upon his subterranean water journey in real fact. Nearly all the river is one vast resonator; its branching avenues and side crevices, its lofty roof of limestone rock, its ancient shores all serve as reflectors of every sound. They send it back intensified a thousand times, blended into one sweet volume of glorious harmony. Nowhere on earth, or in it, can such rich tones be heard, coming back with ever-diminishing volume as they roll down along the unknown halls and are reflected from secret chambers.

Far within the great cavern occur many interesting and fantastic groupings of stalactite matter that with but little aid from their imagination seem to simulate familiar objects. Near the end of the remarkable Pass of El Ghor, a tortuous, narrow, but lofty channel which marks the work of one of the latest of the underground streams, the walls above and on each side are one indescribable maze of calcite accumulations.

Perhaps visitors to Mammoth Cave are most impressed with the lofty domes and deep pits which are found in some portions of this underground domain. Of those that are accessible to the visitor, the best known are Gorin's Dome, the Bottomless Pit, Mammoth Dome, Napoleon's Dome, the Maelstrom, Scylla and Charybdis and the Star Chamber.

The Star Chamber is a delightful scenic spectacle. Its high dome towering above a semi-circular rock formation forming natural seats is encrusted with minerals reflecting the light from torches in such a manner as to give the impression of a gorgeous star-lit night. The various pits and domes are illuminated by the guides from advantageous points throwing into relief formations white as alabaster, fluted and folded in a thousand curious shapes.

Not the least of the pleasures accompanying a trip through Mammoth Cave is the telling of the many legends and the wealth of tradition by the guides, who serve years of apprenticeship before they are entrusted with the task of piloting parties through the tortuous avenues of the cavern. The work of many brave men went into the exploration of Mammoth Cave before its secrets were uncovered for the visitor. Unknown dangers stalked the first explorers who had nothing to guide them in their trips into strange avenues and queer halls where deep pits might at any time swallow them up. These explorations are still going on and as a matter of fact by the descendants of the men who first undertook the work. Some families have carried on the work of exploring the cave for three generations, demonstrating the fascination which attracts these people to this most unusual occupation. The visitor need have no fear for his safety in the cave. Everything has been done which will protect the traveler through Mammoth Cave. Strong bridges have been thrown across the yawning pits, level boardwalks skirt the otherwise hazardous cliffs surrounding such places as the Dead Sea or the River Styx, an idea of which can be gleaned from the names by which they are known.

Fanciful flower gardens are found here deep in the earth, not such flowers as are known above the ground, but duplications in rock so real as to be startling. Within Crystal Avenue there are wonderful acicular crystals of gypsum spread out into a thousand fantastic forms that simulate the flowers of the upper earth, but whose petals are gigantic in comparison.

It is impossible to mention, less possible to describe, all the objects of interest to visitors in this most gigantic cavern in the world. It is entirely different from ordinary things. It is not only a trip to another con-

tinent; it is a trip to another world where the imagination has free rein and where the ties of every day existence are lost in flights of fancy. Delicate fairy-like scenes, somberly gloomy vistas, brilliant gem encrusted walls, mysterious recesses hiding flashing waterfalls, either real or in stone, weird views rivaling any scene depicted by Poe or Dante, gorgeously colored formations, contrast and blend and follow in such rapid succession as to stun the senses with the scenic feast offered.

Such are the things which those attending the meeting of the Southern Medical Association can see at Mammoth Cave when they make the trip to Louisville in November. It is a rare opportunity to attend the meeting and at the same time travel to a scene of unrivalled natural beauty. Any traveler who wishes to make complete his record of experiences must visit Mammoth Cave before he can say he has seen all the wonders of the modern world.

Book Reviews

Diseases of the Stomach: A Textbook for Practitioners and Students. By Max Einhorn, M.D., Emeritus Professor of Medicine at the New York Post-Graduate Medical School and Hospital; Consulting Physician to the Lenox Hill Hospital, New York. New York: William Wood & Co., 1930.

Just fresh from the press, the seventh edition of "Diseases of the Stomach," by Dr. Einhorn, is hailed by practitioners and students with the same enthusiasm which has been accorded all the medical writings of the real father of American gastro-enterology. In his seventh edition Dr. Einhorn has revised the text and brought it up to the minute, so that this volume may be considered the last word to date on diseases of the stomach. He has dealt with the subject from a practical, clinical standpoint.

It is noted that the chapter on functional diseases holds the same relative prominence as in the former editions, which signifies that the author, like many of his colleagues, believes that there are disorders of the stomach which have no recognizable pathologic-anatomic basis, and which yet give a definite and more or less constant symptom-complex, and, therefore, should be given a definite place in textbooks dealing comprehensively with diseases of the alimentary tract.

In Chapter II, Dr. Einhorn takes up the vital subject of examination of the patient. He starts out, as any experienced clinician does, at the bedside of his patient, with the interrogation of the patient. He then goes on to give a graphic description of all the accepted methods of examination of the stomach. The various instruments are pictured, and in some instances the method of applying them is shown by illustrations. The important subject of diet in health as well as in diseased conditions of the stomach, is dealt with in a broad, clear cut way, and forms one of the most important sections of this useful work.

William Wood & Company have exercised the same skill and good judgment in making up this volume which has characterized their publications for half a century and more. The author and the publishers of the seventh edition of Einhorn's masterly work have every reason to be gratified over this last joint effort.

General Surgery. Edited by Evarts A. Graham, A.B., M.D., Professor of Surgery, Washington University School of Medicine, Surgeon-in-Chief of the Barnes Hospital and the Children's Hospital, St. Louis. Series 1929. Volume of the Practical Medicine Series, the Year Book Publishers, 1930.

This is a digest of the world's surgical literature for the past year. An article of unusual significance is a report on the use of the electro-surgical unit of Bovie in the treatment of brain tumors, by Harvey Cushing. Barr, Bulger and Dixon contribute a report on hyperparathyroidism associated with adenoma of the parathyroid gland. Holder recommends a method of anesthesia which lasts five to ten minutes. There are many reports on the use of radium in malignancy this year. The book is replete with discussions of every phase of surgery and will enable one to obtain a group of treatment in line with modern thought.

Treatment in General Practice. By Harry Beckman, M.D., Professor of Pharmacology at Marquette University, Milwaukee, Wisconsin. Octavo volume of 899 pages. Philadelphia and London: W. B. Saunders Company, 1930. Cloth, $10.00 net.

This is a very complete, comprehensive and satisfactory work on treatment, always a dangerous subject to handle. A brief paragraph or two on each disease precedes the therapy and increases the value and interest of the book. These paragraphs include such things as a general description of the disease, variations in virulence, causative factors, incidence and distribution, morbidity and mortality, interesting or unusual features and prognosis, all of which make interesting reading when presented in the author's very readable style. Perhaps it is this latter factor which contributes so much to the value of the book.

The therapy of each disease is given in whatever detail the condition calls for. It includes complete management in general as well as drugs. Prescriptions are given, dosage and intervals. And in many instances the author gives reasons for certain treatment and action of drugs. Dr. Beckman apparently has taken very much to heart the task of supplying the general medical man with a satisfactory work of this kind and he has succeeded remarkably well.

This book is comparable in every way to a recent remarkable work by an English author and carries the added advantage here of using the U. S. P. nomenclature, no small advantage to one who has tried in vain to locate some drug mentioned in the English work.

Otologic Surgery. By Samuel J. Kopetzky, M.D., F.A.C.S., Professor of Otology, Polyclinic Medical School and Hospital, New York; Director, Department of Otolaryngology, Beth Israel Hospital, New York. Second Edition Revised. 553 pages, with 104 illustrations, including 4 color plates; 21 charts. New York: Paul B. Hoeber, Inc., 1929. Cloth, $8.00.

The first edition of this work, which appeared in 1925, was accorded well merited praise.

In the present (second) edition, the subject matter has been brought up to date and the chapter on laboratory aid to diagnosis amplified. Containing, as it does, a complete and detailed discussion of the problems and procedures of otologic surgery, this book should be quite useful to the practising otologist.

Gastric and Duodenal Ulcer. By Arthur F. Hurst, M.A., M.D. (Oxon.), F.R.C.P., Senior Physician to Guy's Hospital, and Matthew J. Stewart, M.B. (Glasg.), F.R.C.P., Professor of Pathology, University of Leeds. With the cooperation in the Radiological Sections of P. J. Briggs, M.A. (Cantab.), M.R.C.S., L.R.C.P., Radiologist to New Lodge Clinic and the Medico-Neurological Clinic at Guy's Hospital. 544 pages, illustrated. New York: Oxford University Press, 1929. Cloth, $20.00.

To those who desire to delve deep into the scientific study of the various phases of gastric and duodenal ulcers the book by Hurst and Stewart will prove of unusual interest, because it is the most thorough and the most complete treatise on the subject that has ever been written. The normal anatomy and the newer physiology of the stomach and duodenum are considered in an introductory chapter. The pathogenesis, the pathological anatomy and the pathological physiology of gastric and duodenal ulcers are discussed in great detail, more than a hundred pages of text, with many beautiful illustrations and colored half tone engravings being given over to those subjects. A most valuable chapter is that on the radiological investigations in ulcer. The x-ray pictures, with accompanying explanatory diagrams, illustrating the various types of, and the changes in, ulcer, are works of art in roentgenography, drawing, and lithographing. The chapters on hemorrhage, perforation, pyloric obstruction and other complications of ulcer are excellent. A disappointing feature of the book is the dismissal of the subject of diet in three scant pages. The authors condemn the schemes of ulcer diets as arranged by von Leube, Lenharz and Sippy as being without rational basis; yet they substitute a less rational plan, giving five ounces of milk every two hours, alternating every other hour with arrow root or purees of potato and cauliflower, for an indefinite period. Olive oil is given four times a day in their "intermediate" ulcer diet. Atropine and the alkalis, tribasic magnesium, calcium phosphate and sodium citrate are the medicines advised. Tobacco is not allowed during the treatment of ulcer.

While the authors believe that surgery is necessary in some cases of ulcer, they assert that medical treatment, if properly carried out, will cure a larger percentage of cases. They, however, discuss at length the indications for, and the advantages and disadvantages of the various ulcer operations. The chapters on the ill results of operations and those on jejunal and gastro-jejunal ulcers will make profitable reading for surgeons, particularly those of the amateur class. The authors and the Oxford Press deserve congratulations on the excellence of this monumental monograph.

Varicose Veins with Special Reference to the Injection Treatment. By H. O. McPheeters, M.D., F.A.C.S., Director of the Varicose Vein and Ulcer Clinic, Minneapolis General Hospital. 233 pages, illustrated with half tone and line engravings. Second Revised and Enlarged Edition. Philadelphia: F. A. Davis Company, 1930. Cloth, $3.50.

This authoritative treatise on the injection treatment of varicose veins is based on a review of the literature and practical experience gained from treating over 800 cases. The rational of the injection method is discussed from an anatomical and physiological viewpoint. Differential diagnosis, treatment and possible complications are given in detail. The book contains an excellent bibliography and can be highly recommended.

Obstetrics. Edited by Joseph B. DeLee, A.M., M.D., Professor of Obstetrics, Northwestern University Medical School, and J. P. Greenhill, B.S., M.D., F.A.C.S., Attending Obstetrician, Chicago Lying-in Hospital and Dispensary. Gynecology, Edited by John Osborn Polak, M.D., Professor of Gynecology, Long Island College Hospital, Brooklyn, New York. A Volume of The Practical Medicine Series, 1928. Chicago: The Year Book Publishers. Cloth, $2.50.

The usual plan of arrangement has been followed in this volume of The Practical Medicine Series of 1928. The fact that Drs. DeLee and Greenhill have edited the division on obstetrics insures a careful evaluation of the newer methods advocated and under discussion. Dr. Polak has edited the section on gynecology. The work of leading men in each field of medicine is abstracted, results are discussed and there is often added a note by the editor. As a supplement to a complete work on either of these subjects it fills a definite place, but it should be accepted as a supplement only. A definite and logical method of arrangement is followed, but naturally the different paragraphs are often not closely related. The book corresponds closely to an encyclopedia, in which the work is arranged according to subject matter instead of alphabetically.

Obstetrics for Nurses. By Joseph B. DeLee, M.D., Professor of Obstetrics and Gynecology, University of Chicago, School of Medicine; Obstetrician to the Chicago Lying-In Hospital and Dispensary. New (9th) Edition, Revised. 12mo. 645 pages, with 269 illustrations. Philadelphia and London: W. B. Saunders Company, 1930. Cloth, $3.00.

In this new edition the same outline is followed as in previous editions. The subject of obstetrical nursing is divided into three parts. The first deals with the anatomy and physiology of reproduction; the second, with labor and the puerperium; and the third, with the pathologies of pregnancy and the new-born child. In addition there is an appendix which discusses allied subjects. The book is well written and goes very much into detail. More space and time are given to operative technic than seem necessary. A welcome note is the stressing of home care. The use of scenes from motion picture films, as well as the numerous illustrations, serve to fix the subject in the mind of the student. The book can readily be employed by the average student, and it is an excellent book for reference.

A Synopsis of Surgery. By Ernest W. Hey Groves, M.S., M.D., B.Sc. (Lond.), F.R.C.S. (Eng.), Surgeon to the Bristol General Hospital; Professor of Surgery, Bristol University; Member of the Court of Examiners, Royal College of Surgeons, England; Examiner in Surgery, Universities of London, Liverpool, Leeds, and Sheffield. Ninth Edition. 676 pages. New York: William Wood & Company, 1930. Cloth, $5.00.

This new, ninth edition of this book brings it up to date and attests its value to the student and practitioner. The subjects of shock, spinal anesthesia, fractures of the neck of the femur, tumors of the bladder and testis and infections of the kidney have been thoroughly revised. The use of radium in malignancy is outlined. Spondylitis, the injection method of the treatment of varices, and the Winnett-Orr method of treating osteomyelitis are given. It is an excellent book for handy reference and for differential diagnosis.

Esophageal Obstruction, Its Pathology, Diagnosis and Treatment. By A. Lawrence Abell, M.S. (Lond.), F.R.C.S. (Eng.), Assistant Surgeon to the Cancer Hospital, London. 234 pages, illustrated. New York: Oxford University Press, 1929. Cloth, $9.00.

This book represents an amplification of a Jacksonian Prize Essay of the Royal College of Surgeons of England, and a Hunterian Lecture, delivered at the Royal College of Surgeons.

It is a very comprehensive and instructive treatise on the subject of esophageal obstruction of all varieties and causes. It presents the opinions and experience, not only of the author, but of all the workers, both European and American, whose researches and contributions have added to our knowledge of esophageal disease. In addition to its own wealth of information, this book possesses the added value of containing an unusually complete bibliography at the end of each chapter, affording a ready reference to the numerous original articles quoted or referred to in the text. It is well illustrated throughout, by diagrammatic drawings, by reproductions of roentgenograms and photographs, and by many excellent colored plates.

The Treatment of Varicose Veins of the Lower Extremities by Injections. T. Henry Treves-Barber, M.D., B.Sc. 120 pages. New York: William Wood & Company, 1929. Cloth, $2.25.

This is an epitome of several years' personal observation and experience in the treatment of varices of the lower extremity. The author classifies varicose veins into three types: primary juvenile, secondary, or acquired and dilated, or compensatory. Distinction between the veins in which the injection method does well and those in which the results are not so pleasing is stressed. Sclerosing agents are discussed in detail. Illustrations are few in number and no bibliography is given. This book is to be recommended as a conservative guide in treating these cases.

Department of Hospitals, City of New York. The Bellevue Hospital Nomenclature of Diseases and Conditions. Revised by the Committee on Clinical Records. Approved by Dr. William Schroeder, Jr., Commissioner, 1929. New York: Paul B. Hoeber, Inc. Cloth, $3.00.

This book provides a standard method for the classification of diagnoses. Especially useful to the hospital, it is applicable to the practitioner's records. It has been completely revised on the basis of new diagnoses and to conform to recent advances in medicine. The cardiac section has been radically changed; in addition to the anatomical classification of the heart, two subdivisions have been added to provide for etiological and physiological diagnoses.

A Textbook of Physiology for Nurses. By William Gay Christian, M.D., Professor of Anatomy, Medical College of Virginia, and Charles C. Haskell, B.A., M.D., Professor of Physiology and Pharmacology, Medical College of Virginia. Second Edition. 153 pages. St. Louis: The C. V. Mosby Company, 1929. Cloth, $2.00.

An elementary textbook suitable for nurses, who are high school graduates. The subject matter is well written, up to date, but rather brief. Laboratory demonstrations and more detailed discussions will be required of the instructor.

Dermatology and Syphilis. Edited by William Allen Pusey, A.M., M.D., Emeritus Professor of Dermatology, College of Medicine, University of Illinois, and Francis Eugene Senear, B.S., M.D., Professor and Head of the Department of Dermatology and Syphilology, College of Medicine, University of Illinois, with the collaboration of Max S. Wien, M.D., Assistant Professor of Dermatology, College of Medicine, University of Illinois. Urology. Edited by John H. Cunningham, M.D., Associate in Genito-Urinary Surgery, Harvard University Post-Graduate School of Medicine. Series 1929. Volume of the Practical Medicine Series. Chicago: The Year Book Publishers, 1930. Cloth, $2.25.

This book is divided into two parts, the one covering the literature of dermatology and syphilis, the other urology. Much experimental work has been done on eczema, both as to etiology and treatment. Many causes of urticaria are given. Attention is given to the frequency of tularemia and the fact that animals other than rabbits may carry the disease. The latest methods of treatment are given. An intensely interesting article on syphilis by E. Brunsgoord tells of end-results of a series of patients of the late Cesar Boeck. The Hinton glycero-cholesterol reaction for diagnosis is discussed.

In the field of urology there are many papers on the newer urinary antiseptics, with a tendency toward conservatism. Many unusual cases and anomalies are reported. A marked advance in diagnostic methods is the use of intravenous pyelography. The work will enable the practitioner to fill out his knowledge of these specialized fields.

Venereal Disease, Its Prevention, Symptoms and Treatment. By Hugh Wansey Bayly, M.C., Hon. Sec. Society for the Prevention of Venereal Disease. 242 pages, with 3 colored plates and 74 illustrations. Fourth (American) Edition. Philadelphia: F. A. Davis Company, 1930. Cloth, $3.50.

This is a concise presentation of the essentials of venereal disease prevention, diagnosis and treatment. It is divided into five sections. The first reviews the different methods of prevention, such as education, legislation and prophylaxis; the second gives the diagnosis and treatment of lues; the third, gonorrhea, both male and female; the fourth, non-specific lesions and the last section discusses the patient. The book is well written and clear in outline. The methods are practical, thorough and up to date.

General Therapeutics. By Bernard Fantus, M.S., M.D., Associate Clinical Professor of Medicine, Rush Medical College of the University of Chicago; Member, Revision Committee United States Pharmacopoeia and of National Formulary Revision Committee. Series 1929. 440 pages. Chicago: The Year Book Publishers. Price of this volume, $2.25.

This is a resume of the progress of therapeutics during the past year. Of particular interest to the Southern practitioner are the discussion of the value of plasmochin in the treatment of malaria, the administration of chinoform (yatren, 105) in amebic dysentery and further studies in the use of oil of chenopodium as an anthelmintic. Of more general interest are the articles on the injection treatment of varices, spinal anesthesia, the evaluation of ultraviolet therapy and the tendency toward smaller doses in the administration of the arsenicals.

Ultraviolet Rays in the Treatment and Cure of Disease. By Percy Hall, M.R.C.S. (Eng.), L.R.C.P. (Lond.), Hon. Actino-Therapist, the Mount Vernon Hospital, London and Northwood. With introductions by Sir Henry Gauvain, M.A., M.D., M.C. (Camb.), F.R.C.S., Medical Superintendent, Lord Mayor Treloar Cripples' Hospitals, and Leonard E. Hill, M.B. (Lond.), F.R.S., Director, Department of Applied Physiology and Hygiene National Institute of Medical Research, London. Fourth Edition. 248 pages. St. Louis: The C. V. Mosby Company, 1930. Cloth, $4.50.

A discussion of the physics of radiation, with its physiological and biological reaction on the human organism is given in the first few chapters of this book. Many models of the different types of lights are illustrated and their advantages given. The remaining chapters concern themselves with the treatment of different conditions, as well as giving case histories in support of the claims. This book will give the general man some grounding in the use of actinotherapy, though the author's reports of results are somewhat enthusiastic.

The Improved Prophylactic Method in the Treatment of Eclampsia. By Prof. W. Stroganoff, Honorary Fellow of the Royal Academy of Medicine in Ireland and Obstetrical-Gynecological Societies of Belgium, Edinburgh, Leningrad, and Moscow. Third Edition, Thoroughly Revised and Completed. First English Edition. 154 pages. New York: William Wood & Co., 1930. Cloth, 3.50.

The first English Edition of the "Improved Prophylactic Treatment of Eclampsia" contains a review of the methods now in vogue and sets forth, by means of case histories and statistics, the advantages of Stroganoff's method, which is outlined in detail.

Procedure in Examination of the Lungs, with Special Reference to the Diagnosis of Tuberculosis. By Arthur F. Kraetzer, M.D., Chief, Medical Department, New York Skin and Cancer Hospital. With a foreword by James Alexander Miller, M.D. 125 pages. New York: Oxford University Press, 1930.

The procedure of lung examination is explained in detail, stressing the early diagnosis of tuberculosis. An old subject is treated with originality and from a practical viewpoint for the medical student.

Roentgenographic Technic. A Manual for Physicians, Students, and Technicians. By Darmon Artelle Rhinehart, A.M., M.D., Professor of Roentgenology and Applied Anatomy, School of Medicine, University of Arkansas. 388 pages, with 159 illustrations. Philadelphia: Lea and Febiger, 1930. Cloth, $5.50.

There is, at present, a trend toward too much diversity in x-ray technic and the author of this book has written it with the idea of presenting a standard to be used, with modification, by technicians. It is practical and up to date; and obsolete methods have been omitted. The illustrations are well chosen and directions for carrying out technic clearly defined. The examination of the sternum, as a rule, is not well done by the average technician, but if one follows the directions given in this book, good results should be expected. The formulae for x-ray exposure of the different anatomical regions of the body should be a great advantage to technicians.

Applied Physiology. By Samson Wright, M.D., M.R.C.P., Lecturer in Physiology, University of London, King's College. With an Introduction by Swale Vincent, M.D., Professor of Physiology, University of London. Third edition, with 550 pages. Oxford University Press, New York, 1929.

The facts presented are accurate and the newest physiological concepts are included. In the discussion of recent discoveries, American work perhaps does not receive as much recognition as it deserves. For example, Corner, Allen, Doisy, and Frank's names are not mentioned in the account of ovarian hormones. The bibliographical references in general are limited.

Not for its charm of expression, but as a reference book, briefer than the average text of physiology, and as an authoritative review of present day knowledge, it apparently leaves nothing to be desired.

Southern Medical News

ALABAMA

At the annual meeting of the physicians and surgeons representing the Alabama Power Company and the Southern Division of Allied Engineers, held recently at Mitchell Dam, Dr. J. S. Turberville, of Century, Florida, was elected President, and Dr. W. M. Gamble, of Wetumpka, Vice-President. Mitchell Dam was chosen for future meetings. Dr. William Chaney, of Memphis, Tennessee, was awarded the annual prize of the Association for the best paper read.

The Chattahoochee Valley Medical and Surgical Association will meet in Albany, Georgia, July 8-9, under the Presidency of Dr. Henry Green, of Dothan. A number of Alabama physicians and surgeons will read papers.

Reports from the State Health Department of Alabama show an increase in malaria from 11.4 per hundred thousand of population in 1928 to 16.6 in 1929, and in influenza from 72.3 in 1928 to 121.3 in 1929. Heart disease was the chief cause of death with a percentage of 136.3 per hundred thousand of population.

Dr. W. W. Harper, Selma, has been elected Recording Secretary of the Association of Surgeons of the Southern Railway System, and Dr. C. N. Parnell, Maplesville, Second Vice-President.

Deaths

Dr. Josian Edward Evans, Mobile, aged 60, died February 25, of atrophic cirrhosis of the liver.

ARKANSAS

The Arkansas Medical Society, at its annual meeting held in Fort Smith, May 6-8, elected Dr. Edward E. Barlow, Dermott, President; Dr. Darmon A. Rhinehart, Little Rock, President-Elect; Dr. William H. Mock, Prairie Grove, Dr. Henry B. Hardy, Greenbrier, and Dr. James B. Ellis, Helena, Vice-Presidents; Dr. William R. Bathurst, Little Rock, Secretary, reelected; and Dr. Royal J. Calcote, Little Rock, Treasurer.

Crawford County Medical Association has elected Dr. J. M. Stewart, Van Buren, President; Dr. Q. R. Galloway, Mulberry, Vice-President; Dr. Odell J. Kirksey, Mulberry, Secretary-Treasurer.

Dr. B. A. Rhinehart, Little Rock, won the Dewell Gann Cup, given for the high score in golf, at the recent annual meeting of the Arkansas Medical Society in Fort Smith.

Dr. Allan A. Gilbert and Miss Esther Fenlon, both of Fayetteville, were married in Fort Smith, May 7.

Deaths

Dr. James Leon Greene, Hot Springs, aged 68, died April 21 of heart disease.

Dr. John Lewis Smiley, Siloam Springs, aged 55, died April 15 of angina pectoris.

Dr. Horace F. Villars, Little Rock, aged 60, died April 24.

DISTRICT OF COLUMBIA

The Medical Society of Virginia, Maryland and District of Columbia, recently held its semi-annual meeting with Dr. Wm. T. Davis as President; Dr. Robert Scott Lamb, as Treasurer; and Dr. Joseph D. Rogers as Corresponding Secretary, all of Washington.

Dr. George R. Callender, Washington, was elected President of the American Association of Pathologists and Bacteriologists at the recent meeting of this Association held in New York.

Major William E. M. Devers has been relieved from his duties in the Surgeon General's Office at Washington and assigned to duty at St. Louis Medical Depot.

Major Edward A. Noyes has been assigned to duty in the Surgeon General's Office at Washington, having been relieved from duty at Letterman General Hospital, San Francisco.

Deaths

Dr. Joseph D. Blair, Washington, aged 63, died March 21, of arteriosclerosis and chronic nephritis.

Dr. Patrick James Byrne, Washington, aged 76, died recently.

Dr. John Parsons, Washington, aged 83, died February 10, of arteriosclerosis and myocarditis.

Dr. Harry Martin Price, Washington, aged 55, died April 8, of pneumonia.

FLORIDA

At the meeting of the Florida State Medical Association held recently in Pensacola, Dr. Julius C. Davis, of Quincy, was elected President; Dr. Walter C. Payne, Pensacola, Dr. William H. Spiers, Orlando, and Dr. Mathew J. Flipse, Miami, Vice-Presidents; Dr. Shaler Richardson, Jacksonville, Secretary, reelected. Orlando was chosen for the 1931 meeting place.

Washington-Holmes County Medical Society has elected Dr. Wm. C. Coleman, Chipley, President; Dr. H. A. McClure, Chipley, Secretary-Treasurer.

Drs. J. M. Whitfield and Don S. Fraser, of Panama City, are erecting a thirty-bed hospital there. These physicians will take care of the employees of the International Paper Company.

The East Coast Medical Association will hold its next meeting at Melbourne, October 2 and 3.

Dr. E. Gordon Fletcher, of Oakland, California, was recently appointed Assistant Chief Surgeon of the Florida East Coast Railway Company and East Coast Hospital Association at St. Augustine.

Dr. C. H. Farmer, of Lakeland, did special work at the St. Louis Children's Hospital, St. Louis, Missouri, recently. Beginning with July he will be connected with the Infants' and Children's Hospital at Saluda, North Carolina.

Dr. Leland Hargreaves Dame has moved to Inverness to be associated with his brother, Dr. George A. Dame.

Dr. and Mrs. J. L. Kirby-Smith, of Jacksonville, recently visited his brother in Tuincucu, Cuba. Dr. Kirby-Smith has recovered from an illness of several months and has resumed his professional duties.

Dr. John S. Helms, of Tampa, will present a paper on "The Incidence of Goiter in the Southeastern States and Its Possible Significance" at the meeting of the American Association for the Study of Goiter to be held in Seattle, Washington, July 10 and 11.

Dr. Carl Williams, of St. Petersburg, has been appointed a member of the State Board of Medical Examiners by the Governor.

The Children's Home Society of Florida, Jacksonville, has elected Dr. Luther W. Holloway a member of the Board of Directors and Chief of Staff to take the place of the late Dr. James D. Love. Other members of the staff are: Medicine—Dr. R. H. McGinnis, Dr. T. Z. Cason, Dr. Herman H. Harris, Dr. T. E. Buckman and Dr. Thomas Morgan; Surgery—Dr. Edward Jelks, Dr. J. Knox Simpson, Dr. H. A. Peyton and Dr. James Hartman; Gynecology—Dr. R. B. McIver; Eye, Ear, Nose and Throat—Dr. H. Marshall Taylor, Dr. N. M. Heggie, Dr. Shaler Richardson and Dr. William Knauer; Roentgenology—Dr. L. W. Cunningham and Dr. W. M. Shaw; Neurology—Dr. Ralph N. Green; Dermatology—Dr. J. L. Kirby-Smith; Urology—Dr. Thomas Thompson and Dr. E. T. Sellers.

Dr. R. C. Woodard, Miami, has moved his offices from the Venetian Building to 27 N. W. Twelfth Avenue.

Deaths

Dr. W. A. Oughterson, Palm Beach, aged 55, died March 28, of cerebral embolus.

Dr. C. Eugene Riggs, of St. Paul, Minnesota, who has spent winters in Florida for many years, died April 4.

Dr. Arthur W. Underwood, St. Augustine, aged 64, died April 1.

GEORGIA

The Georgia State Medical Association at its recent annual meeting elected Dr. G. Y. Moore, Cuthbert, President; Dr. A. G. Fort, Atlanta, President-Elect; Dr. Geo. A. Traylor, Augusta, 1st Vice-President; Dr. S. T. R. Revell, Louisville, 2nd Vice-President; Dr. Allen H. Bunce, Atlanta, Secretary-Treasurer, re-elected. Atlanta was chosen for the 1931 meeting place.

Butts County Medical Society has elected Dr. A. F. White, Flovilla, President; Dr. O. B. Howell, Jackson, Vice-President; Dr. R. L. Hammond, Jackson, Secretary-Treasurer.

Baldwin County Medical Society has elected Dr. John W. Mobley, Jr., Milledgeville, President; Dr. C. G. Cox, Milledgeville, Vice-President; Dr. John D. Wiley, Milledgeville, Secretary-Treasurer.

Crisp County Medical Society has elected Dr. Guy G. Lunsford, Cordele, President; Dr. Charles Adams, Cordele, Vice-President; Dr. J. N. Dorminy, Cordele, Secretary.

Cobb County Medical Society has elected Dr. W. M. Gober, Marietta, President; Dr. R. W. Fowler, Marietta, Vice-President; Dr. L. L. Welch, Marietta, Secretary-Treasurer.

Colquit County Medical Society has elected Dr. J. J. C. Wright, Doerun, President; Dr. E. L. Lawson, Moultrie, Vice-President; Dr. T. H. Chestnutt, Moultrie, Secretary-Treasurer.

Franklin County Medical Society has elected Dr. Stewart D. Brown, Royston, President; Dr. B. T. Smith, Carnesville, Secretary-Treasurer.

Glynn County Medical Society has elected Dr. H. L. Akridge, Brunswick, President; Dr. H. M. Branham, Brunswick, Vice-President; Dr. F. N. Aldrich, Brunswick, Secretary-Treasurer.

Hancock County Medical Society has elected Dr. Horace Darden, Sparta, President; Dr. H. L. Earl, Sparta, Secretary-Treasurer.

Laurens County Medical Society has elected Dr. J. J. Barton, Dublin, President; Dr. C. A. Hodges, Dublin, Vice-President; Dr. O. H. Cheek, Dublin, Secretary-Treasurer.

Stephens County Medical Society has elected Dr. W. H. Swain, Martin, President; Dr. J. H. Terrell, Toccoa, Vice-President; Dr. C. L. Ayers, Toccoa, Secretary-Treasurer.

Taliaferro County Medical Society has elected Dr. John A. Rhodes, Crawfordville, President; Dr. A. T. Ray, Sharon, Secretary-Treasurer.

Troup County Medical Society has elected Dr. Enoch Callaway, LaGrange, President; Dr. S. C. Rutland, LaGrange, Vice-President; Dr. H. McCulloh, Jr., West Point, Secretary.

Upson County Medical Society has elected Dr. D. W. Pritchett, Thomaston, President; Dr. F. M. Woodall, Thomaston, Vice-President; Dr. R. L. Carter, Thomaston, Secretary-Treasurer.

Walton County Medical Society has elected Dr. H. L. Upshaw, Social Circle, President; Dr. J. B. H. Day, Social Circle, Vice-President; Dr. J. K. McClintic, Monroe, Secretary-Treasurer.

Wayne County Medical Society has elected Dr. J. Lawton Tye, Screven, President; Dr. I. K. Ogden, Odum, Vice-President; Dr. A. J. Gordon, Jesup, Secretary-Treasurer.

Whitfield County Medical Society has elected Dr. R. S. Bradley, Dalton, President; Dr. J. C. Rollins,

(Continued on page 34)

for

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TO rid the malarial patient of infection it is essential to destroy both the schizonts which are chiefly responsible for the attacks and the gametes or sexual forms which transmit the disease through the mosquito. While quinin is destructive to the former, it has but a limited action upon the latter. For this reason chemists and research investigators have long sought an antimalarial agent that would be effective against all types of plasmodia. This aim has been finally attained by

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(Continued from page 680)

Dalton, Vice-President; Dr. E. O. Shellhorse, Dalton, Secretary-Treasurer.

The first annual meeting of the Southeastern Surgical Association, recently organized, was held in Augusta at the time of the State Medical Association meeting. The new officers elected are: Dr. E. G. Ballenger, President; Dr. C. W. Roberts, President-Elect; Dr. L. C. Fischer, Vice-President; Dr. B. T. Beasley, Executive Secretary and Director-General. Dr. D. Y. Sage and Dr. M. C. Pruitt were elected members of the Executive Council for a period of five and four years, respectively. All of the officers reside in Atlanta. It is contemplated this organization will have its own official publication.

The Georgia Urological Association was organized at the recent meeting of the Medical Association of Georgia. Dr. James N. Brawner, Atlanta, President of the Fulton County Medical Society, presided.

Dr. Herschel Crawford, Atlanta, has returned from Europe where he spent several months doing post-graduate work.

Dr. N. M. Owensby, Atlanta, spent six weeks in the East visiting clinics in New York City, Boston, Philadelphia, Washington and Baltimore.

Dr. B. H. Minchew, Waycross, was the principal speaker at a luncheon meeting of the City Club at the Piedmont Hotel, Atlanta, during April.

Dr. Stewart Roberts, Atlanta, read a paper before the National Tuberculosis Association at the annual meeting held in Memphis, Tennessee, in May.

The Woman's Auxiliary of Georgia has elected Mrs. C. C. Harold, Macon, President, and Mrs. Ralston Lattimore, Savannah, President-Elect.

Dr. Thomas Jackson Charlton, Savannah, and Miss Anne Carrington Read were married April 5.

Dr. Hervey Milton Cleckley and Mrs. Louise Martin Marshall, both of Augusta, were married recently.

Deaths

Dr. William Linwood Harvey, Bartow, aged 41, died April 3.

Dr. Thomas J. Moss, Atlanta, aged 74, died April 28.

Dr. Herbert Rushin, Macon, aged 45, died suddenly March 31 of cerebral hemorrhage.

Dr. William Pellham Turk, Atlanta, aged 60, died February 28, of acute myocarditis and nephritis.

KENTUCKY

Ballard County Medical Society has elected Dr. G. E. Titsworth, Jr., Bandana, President; Dr. W. A. Ashbrook, Gage, Vice-President; Dr. G. L. Thompson, Wickliffe, Secretary.

Franklin County Medical Society has elected Dr. John Patterson, Frankfort, President, reelected; Dr. A. M. Jackson, Frankfort, Vice-President; Dr. L. T. Minish, Frankfort, Secretary-Treasurer.

Henderson County Medical Society has elected Dr. Silas Griffin, Henderson, President; Dr. J. O. Strother, Henderson, Vice-President; Dr. Peyton Ligon, Secretary-Treasurer.

(Continued on page 36)

MALARIA
AND INTERMITTENT FEVERS

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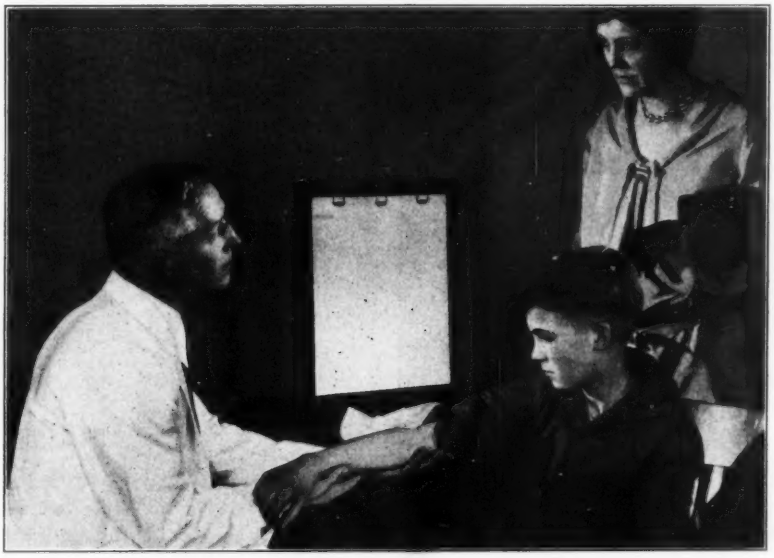

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(Continued from page 34)

Owen County Medical Society has elected Dr. J. H. Chrisman, Owenton, President; Dr. J. W. Botts, Owenton, Vice-President; Dr. K. S. McBee, Owenton, Secretary and Treasurer.

The School of Public Health, University of Louisville, and the State Board of Health, under the direction of the Dean, Dr. A. T. McCormack, State Health Officer, Louisville, are offering an eight months' course in laboratory technique, the next term beginning September 15. Those interested should write to Dr. L. H. South, School of Public Health Laboratory Technique, 532 West Main Street, Louisville, Kentucky.

The Kentucky Hospital Association met April 14 and 15, Dr. John R. Wathen, First Vice-President, presiding at the first session. Dr. C. H. Harris, Director of Health, Louisville, delivered the address of welcome.

Dr. E. H. Schultz, Middlesboro, has been elected Fourth Vice-President of the Association of Surgeons of the Southern Railway System.

Dr. J. Murray Kinsman announces his association with Drs. J. A. and Morris Flexner, Heyburn Building, Louisville.

Deaths

Dr. Charles D. Almon, Cleaton, aged 61, died March 31 at Owensboro, of meningitis as the result of septic tonsils.

Dr. Moses Adkins, Millard, aged 60, died February 22.

Dr. Joseph Ferguson Jones, Mount Sterling, aged 77, died April 18, of heart disease.

Dr. William C. Keen, Burkesville, aged 76, died April 1, of heart disease.

Dr. Algan T. Knox, Thomson, aged 65, died March 26, of cerebral hemorrhage.

Dr. Thomas L. Menaugh, Harrodsburg, aged 69, died March 31, of heart disease.

Dr. Eugene Banks Pendleton, Owensboro, aged 64, died March 9, of chronic myocarditis.

Dr. Alexander Shively, Campbellsville, aged 92, died in March, of senility.

Dr. Isaac Leonard Wyatt, aged 59, died April 25, of an accidental gunshot wound.

LOUISIANA

The Louisiana State Medical Society at its annual meeting held recently elected Dr. Herman B. Gessner, New Orleans, President; Dr. Sidney C. Barrow, Shreveport, President-Elect; Dr. Sambola J. Couvillon, Moreauville, First Vice-President; Dr. Carl A. Weiss, Baton Rouge, Second Vice-President; Dr. Armand G. McHenry, Monroe, Third Vice-President; Dr. Paul T. Talbot, New Orleans, Secretary-Treasurer, reelected. The next annual meeting will be held in New Orleans.

The Louisiana Hospital Association elected the following officers: Dr. E. L. Sanderson, Shreveport, President; Dr. W. C. Rucker, New Orleans, Vice-President; Dr. Arthur Vidrine, New Orleans, Secretary-Treasurer. Baton Rouge was chosen for the next meeting place in April, 1931.

Dr. Herbert Randolph Unsworth has been appointed physician for the Louisiana Retreat, New Orleans, succeeding his father, Dr. Charles V. Unsworth.

Dr. Basil C. MacLean, Montreal, Canada, has been appointed Superintendent of the Touro Infirmary, New Orleans, succeeding the late Dr. Paul W. Wipperman.

Deaths

Dr. Robert Hodges Blackman, Shreveport, aged 59, died April 4, of acute pancreatitis and gastroduodenitis.

Dr. Herbert Claiborne Cole, Monroe, aged 46, died April 7, in Baltimore, Maryland, of heart disease.

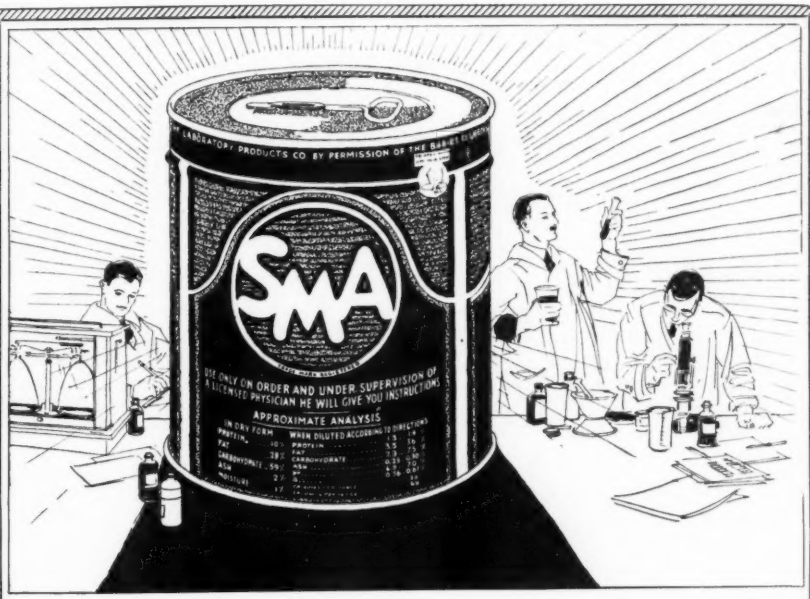
Dr. Carlton Daniel Haas, New Orleans, aged 55, died April 12, of hypertrophy of the prostate and uremia.

Dr. Frederick R. Tolson, Lafayette, aged 77, died March 31, of lobar pneumonia.

MARYLAND

At the recent meeting of the Medical and Chirurgical Faculty of Maryland, Dr. James M. H. Rowland was

(Continued on page 38)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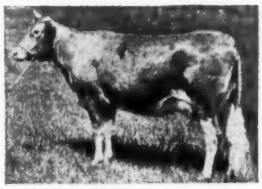

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(Continued from page 36)

elected President; Dr. William D. Campbell, Hagerstown, Dr. Henry M. Landford, Princess Anne, and Dr. Charles W. Maxson, Baltimore, Vice-Presidents; Dr. Charles E. Brack, Baltimore, Treasurer, and Dr. John T. King, Jr., Baltimore, Secretary, reelected.

The Johns Hopkins Hospital and School of Medicine has received from the General Education Board of New York $500,000 to be used in the completion of the William Osler Medical Clinic and the William Stewart Halstead Surgical Clinic. This gift raises the total given by the Board toward the completion of these buildings to $1,750,000. When finished these two buildings will add 165 beds to the hospital. The Osler Clinic is expected to be completed by December, when work will be begun on the Halstead Clinic.

Dr. Frederick H. Baetjer, Professor of Clinical Roentgenology, Johns Hopkins University, School of Medicine, Baltimore, has undergone his fifty-second operation necessitated by injuries from exposure to x-rays.

Dr. Ralph G. Beachley has been appointed full-time Health Officer for Kent County, with headquarters at Chestertown.

In recognition of his fifty years of service as a Medical Examiner of the Baltimore and Ohio Railroad, Dr. James D. Iglehart was presented with a gold watch by his associates. He is still in active practice at the age of 80 years.

Dr. J. Whitridge Williams and Mrs. C. DeW. Theobald Pennington, both of Baltimore, were married April 30.

Deaths

Dr. Milo H. Sutliff, Chevy Chase, aged 64, died April 3, at Washington, D. C., of heart disease.

MISSISSIPPI

The Mississippi State Medical Association, at its recent meeting, elected Dr. E. F. Howard, Vicksburg, President, and Dr. J. C. Culley, Oxford, President-Elect.

The next meeting of the North Mississippi Six County Medical Society will be held in Water Valley in July.

The Woman's Auxiliary of the Mississippi State Medical Association elected the following officers: Mrs. L. L. Polk, Purvis, President; Mrs. G. D. Mason, Lumberton, President-Elect; Mrs. Augustus Street, Vicksburg, First Vice-President; Mrs. W. C. Poole, Cary, Second Vice-President; Mrs. Henry Boswell, Sanatorium, Recording Secretary; Mrs. E. C. Parker, Gulfport, Treasurer; Mrs. D. J. Williams, Gulfport, Parliamentarian.

Deaths

Dr. J. Augustus Evans, Houston, aged 68, died February 11.

MISSOURI

Missouri State Medical Association at its recent meeting elected Dr. C. D. Humberd, Barnard, President; Dr. Howard B. Goodrich, Hannibal, Vice-President; Dr. J. T. Hornback, Nevada, Secretary, reelected.

The new $1,000,000 Jewish Hospital, Kansas City, is now under construction. The normal capacity of the hospital will be 129 beds, but the facilities can be increased to 150.

An institution, which will be known as the Dan and Blanche Bordley Memorial for Chronic Invalids, has been provided for in the will of Mrs. Blanche Bordley, widow of Daniel C. Bordley, formerly a Director of the Liggett & Myers Tobacco Company. The amount of $435,000 has been set aside for the institution.

In conjunction with the ninth annual scientific session of the American Congress of Physical Therapy, to be held in the Hotel Jefferson, St. Louis, September 8 to 12, a postgraduate week of physical therapy will be conducted.

The following officers were elected at the annual

(Continued on page 40)

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(Continued from page 38)

meeting of the staff of the St. Joseph's Hospital, St. Joseph: Dr. John I. Byrne, President; Dr. C. S. Branson, Vice-President; Dr. L. H. Fuson, Treasurer; Dr. G. A. Lau, Secretary.

Dr. Joseph Erlanger, Professor of Physiology in Washington University School of Medicine, St. Louis, delivered the Hitchcock lectures at the University of California in April.

The State Federal Soldiers' Home of Missouri, St. James, was recently destroyed by fire, the loss being estimated at $100,000. Fifty-eight patients in the hospital, most of them unable to walk, were rescued without injury. The House Surgeon, Dr. W. H. Breuer, secured accommodations for the patients in temporary quarters in other buildings on the grounds.

Dr. Alonzo R. Kieffer, a practicing physician in St. Louis for more than fifty years, was honored by the St. Louis Medical Society at its meeting in April, when a bronze plaque bearing the profile of Dr. Kieffer was presented to the Society. It will be hung in the corridors of the Society's headquarters.

The Department of Ophthalmology of Washington University School of Medicine, St. Louis, announced the following appointments: Dr. George H. Bishop, Professor of Applied Physiology; Dr. Louis A. Julianelle, Associate Professor of Applied Bacteriology and Immunology; Dr. James A. Hawkins, Associate Professor of Applied Biochemistry; Dr. R. Wendell Harrison, Instructor in Applied Bacteriology and Immunology; and Dr. Garvey Bowers, Research Assistant in Applied Bacteriology. Dr. Harvey J. Howard is head of the Department of Ophthalmology.

Ground was broken for the projected $200,000 Mothers' and Babies' Home at Ferguson on May 11. This institution is supported by Christian Churches, the Christian Women's Benevolence Association and free-will contributions of individuals. A campaign to secure a $200,000 building fund was launched in June in St. Louis and St. Louis County.

Dr. Benjamin A. Wilkes, Superintendent of the Missouri Baptist Hospital, St. Louis, for thirty-four years, has been granted a leave of absence to take charge of the Hollywood Hospital, Hollywood, California.

Dr. Carl Barck, St. Louis, will be one of the speakers at the Congress of Notable Badensians to be held at Karlsruhe, Germany, July 11 to 14.

A plan to establish a widows' fund in connection with the activities of the Missouri State Medical Association is being sponsored by Dr. Frank I. Ridge, of Kansas City.

Dr. A. J. Smith has been appointed Health Officer of the St. Joseph Board of Health.

Dr. G. T. Bloomer has been appointed Police Surgeon at St. Joseph.

Deaths

Dr. David S. Booth, St. Louis, aged 67, died May 16.

Dr. Oscar Herman Elbrecht, St. Louis, aged 53, died May 5, of pneumonia.

Dr. James Alford Glascock, St. Louis, aged 45, was killed March 21.

Dr. Robert D. Haire, Clinton, aged 75, died April 13, of valvular heart.

Dr. Cook Pruitt Gregg, formerly of St. Louis, aged 52, died en route to Los Angeles with a patient, May 1.

Dr. John H. McCaughan, St. Louis, aged 60, died May 2.

Dr. Mary H. McLean, St. Louis, aged 69, died recently.

Dr. Henry W. Westover, St. Joseph, aged 78, died March 30, of pneumonia and uremia.

Dr. Albert Miller Wilson, Kansas City, aged 75, died April 22 of arteriosclerosis.

NORTH CAROLINA

The North Carolina Tuberculosis Association elected Dr. L. B. McBrayer, Southern Pines, Managing Director, and reelected Mrs. Gordon Finger, Charlotte, President. Mrs. Charles R. Whitaker, Southern Pines, retains the office of Vice-President. Dr. R. L. Carlton, Winston-Salem, was elected Representative Director. The Executive Committee is composed of the

(Continued on page 42)

Merrell-Soule Powdered Protein Milk in the dietetic treatment of Summer Diarrheas

Diarrheas and other intestinal disturbances in infants generally show rapid improvement when Merrell-Soule Powdered Protein Milk is used. Pediatrists attribute its successful action in such cases to a combination of the following factors, exclusive with this product.

1. *Removal of whey salts*
2. *High protein content*
3. *Low milk sugar content*
4. *Acidity of not less than* pH = 4.5
5. *Fine division and friability of the curd*

An increased number of pediatricians are turning to Merrell-Soule Powdered Protein milk as the best product of its kind. It gives results. It is absolutely uniform. (Published analyses are strictly adhered to.) Its convenient powdered form makes its preparation simple and easy. It is a product of proven merit.

Literature and samples sent promptly on request.

Merrell-Soule Co., Inc.,
350 Madison Avenue, New York

(Recognizing the importance of scientific control, all contact with the laity is predicated on the policy that KLIM and its allied products be used in infant feeding only according to a physician's formula.)

Merrell-Soule Powdered Milk Products, including Protein Milk, Whole Lactic Acid Milk and Klim, are packed to keep indefinitely. Trade packages need no expiration date.

(Continued from page 40)

officers and Dr. P. P. McCain, Sanatorium, Dr. J. W. Dickey, Southern Pines, and Mrs. Charles E. Platt, of Charlotte. The new directors are: Dr. J. C. Hudson, Greensboro, Dr. J. L. Spruill, Greensboro, Dr. P. A. Yoder, Sanatorium.

The Southern Pediatric Seminar will be held at Saluda July 28 to August 8. Applications may be made to Dr. D. Lesesne Smith, Spartanburg, South Carolina.

Dr. H. M. Vann, Wake Forest, was made President of the Wake Forest Medical Alumni for the next year.

Deaths

Dr. William Paisley Beall, Greensboro, aged 79, died April 26, of injuries received in an automobile accident.

Dr. Henry Irwin Clark, Scotland Neck, aged 73, died April 17, of heart disease.

Dr. Alfred Preston Davis, Winston-Salem, aged 74, died April 11, of arteriosclerosis and cerebral hemorrhage.

Dr. James Franklin Dicks, Stoneville, aged 53, died March 23, of chronic nephritis.

Dr. Oliver G. Falls, Kings Mountain, aged 73, died April 5, of heart disease.

Dr. Timmons Greenberry Hamrick, Shelby, aged 75, died April 15, of chronic myocarditis.

Dr. Owen Smith, High Point, aged 49, died April 26, of pneumonia.

Dr. George S. Watson, Elon College, aged 85, died March 4, of senility,

OKLAHOMA

Noble County Medical Society has elected Dr. D. F. Coldiron, Perry, President; Dr. J. W. Francis, Perry, Secretary.

Dr. R. Q. Atchley, Tulsa, sailed June 6 for London and Berlin to take post graduate work in surgery. He will return about September 1.

Dr. and Mrs. H. C. Antle, Chickasha, recently went to New Orleans, Louisiana, where Dr. Antle attended a post graduate course in skin diseases and electrotherapy at Tulane University.

Dr. J. H. Rucks, Superintendent of the Wesley Hospital, Oklahoma City, has been elected First Vice-President of the Midwest Hospital Association. Dr. Wann Langston, Superintendent of the University Hospital, Oklahoma City, was appointed one of the new Trustees.

Deaths

Dr. S. Hall Kimmons, Tulsa, aged 62, died March 25 of heart disease.

SOUTH CAROLINA

Dr. Charles A. Mobley, of Orangeburg, was elected Vice-President of the South Carolina Medical Association at the recent meeting. Greenville was chosen for the 1931 meeting.

Dr. Roy D. Metz, of Chick Springs Sanitarium, Chick Springs, addressed Furman University at Greer on April 26 and Converse College at Spartanburg on May 3, his subject for both addresses being "Medicine as a Career."

The Medical College of the State of South Carolina, cooperating with the State Medical Association, will give a Post-Graduate Course in Medicine, Pediatrics, and Obstetrics from July 7 to July 12. Five dollars will be charged as a fee for incidental expenses.

Dr. W. H. Carrigan, Summerton, will be the Director of Laboratories at the new hospital in Sumter.

Dr. Lewis Blake, Spartanburg, has been elected First Vice-President of the Association of Surgeons of the Southern Railway System.

Deaths

Dr. Dempsey Jones Hydrick, Orangeburg, aged 53, died February 3.

Dr. William Lawrence Linder, Union, aged 52, died March 11, in a hospital at Columbia of myocarditis and chronic nephritis.

TENNESSEE

Dr. W. S. Leathers, Professor of Preventive Medicine and Dean of the Vanderbilt Medical School, Nashville, has been elected a member of the Board of Scientific Directors of the International Health Division of the Rockefeller Foundation and has accepted.

Dr. E. Dunbar Newell, Chattanooga, has been elected President of the Association of Surgeons of the Southern Railway System.

At the last staff meeting of the Baptist Memorial Hospital, Memphis, Dr. John J. Shea was elected

(Continued on page 44)

EVERY physician reader of this Journal is welcome to a full size tube of this superior Balm for Burns.

Benzocomp is pain-quelling, soothing and healing. The soft, smooth base permits easy and comfortable application. Granulation is not disturbed when dressings are removed.

Write us today for the complimentary tube . . . the evidence which supports our claims.

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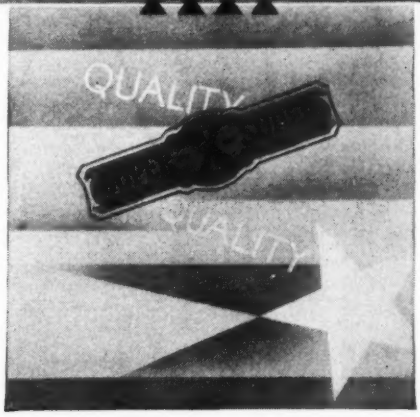

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—in the giving of Greater Values * * a result of Buying Power; FIRST—in providing Selections * * with buyers combing the markets of two hemispheres to insure complete stocks; FIRST—to extend credit through an Easy Rental Purchase Plan ** a result of financial resourcefulness and implicit faith in the integrity of the Profession; FIRST—to organize for Same Day Service * * meeting the modern day demand for speed. And FIRST in point of size—of no importance in itself but significant in that attention to the above factors have made it so. FIRST, really—in Everything * * that's true LEADERSHIP! Proud we are—yet the greatest benefit is yours.

A·S·ALOE COMPANY

CHICAGO ST. LOUIS LOS ANGELES

(Continued from page 42)

President, Dr. Wilson Searight, Vice-President, and Dr. H. K. Turley, Secretary.

Work on the new $2,000,000 medical plant for Meharry Medical College was begun recently. According to the contract, it is to be completed in about sixteen months.

Dr. O. O. Jones, roentgenologist at the Crook Sanatorium at Jackson for the past two years, accepted a position as anesthetist and roentgenologist with a clinic at Houston, Texas.

Dr. Cecil E. Newell, of Chattanooga, has accepted a three-year scholarship at the Mayo Clinic. For the last two years he has been associated with his uncle, Dr. Edward T. Newell.

Dr. W. Gardner Rhea has joined the Wiggins Clinic at Paris.

St. Mary's Memorial Hospital, Knoxville, was formally opened April 22.

Dr. James K. Blackburn, Pulaski, has been reappointed to serve on the State Board of Medical Examiners.

Dr. Andrew F. Richards, Director of the Knox County Health Unit, has resigned to become Associate Director of State Health Units, and he succeeds Dr. Amos G. Hufstedler.

In a check-up of the alumni of Vanderbilt University, Nashville, it was found that of 12,896 living graduates, 4,308 were practicing medicine.

Dr. W. B. Harrison, formerly County Health Officer of Shelby County, has been made Associate Director of the State Department of Public Health for West Tennessee. Dr. W. P. Moore, who was formerly connected with the Shelby County Health Department, succeeded Dr. Harrison as County Health Officer of that county, and is located at Memphis.

Dr. William Anson Garrot, Memphis, and Miss Ruth La Clair Waltrip, Cadiz, Kentucky, were married April 17.

Dr. George W. Webb and Mrs. Louvenie Cook, both of Redboiling Springs, were married April 5.

Deaths

Dr. Kenneth La Fayette Cook, Clifton, aged 77, died April 10 of cerebral hemorrhage.

Dr. Sidney Alonso Moffitt, Cornersville, aged 59, died in April of nephritis.

Dr. Henry Posert, Memphis, aged 71, died March 31, of heart disease.

TEXAS

The State Medical Association of Texas, at its recent annual meeting, elected Dr. John W. Burns, Cuero, President; Dr. John O. McReynolds, Dallas, President-Elect; Dr. William L. Baugh, Lubbock, Dr. Charles W. Stevenson, Wichita Falls, and Dr. Robert B. Bledsoe, Lufkin, Vice-Presidents; and Dr. Holman Taylor, Fort Worth, Secretary, reelected. Beaumont was chosen as the 1931 meeting place.

The Tri-State Medical Society, at its twenty-fifth annual meeting, held at Marshall, elected Dr. J. A. Daniels, Carthage, President; Dr. J. H. Spivey, Henderson, Dr. Edgar Hull, Pleasant Hill, Louisiana, and Dr. Hugo Longino, Texarkana, Arkansas, Vice-Presidents; Dr. Frank Walke, Shreveport, Louisiana, Secretary-Treasurer, reelected. Shreveport, Louisiana, was chosen as the place for the 1931 meeting.

Hidalgo County Medical Society has elected Dr. T. W. Glass, Weslaco, President; Dr. C. J. Hamme, Edinburg, Vice-President; Dr. J. D. Stevens, Weslaco, Secretary-Treasurer.

Parker County Medical Society has elected Dr. M. Thompson, President; Dr. Phil R. Simmons, Vice-President; Dr. Alexander S. Garrett, Secretary-Treasurer, reelected.

Wood County Medical Society has elected Dr. Robert H. Coleman, Mineola, President; Dr. T. B. Reed, Mineola, Vice-President; Dr. J. M. Puckett, Mineola, Secretary-Treasurer.

(Continued on page 46)

A Dependable Agent for Supplying Calcium in Diffusible Form

KALAK WATER presents a saturate solution of calcium as the bicarbonate.

"Of the inorganic salts of calcium, the bicarbonate was the most effective in raising the blood calcium." (W. H. Jansen—Deut. Arch. f. klin Med. Oct. 1924.)

It is not possible to present calcium bicarbonate as a dry powder or as an effervescent mixture to be added to water. This salt can be maintained as the bicarbonate only in an aqueous solution that is charged with carbon dioxide.

KALAK WATER CO.
6 Church Street » » New York City

Patient Types . . .

The Expectant Mother

DURING the anxious period of pregnancy, you are "the law and the prophets" to the woman. Sympathy and understanding incline you to select a smooth, palatable and comfortable aid to the essential normal peristalsis. What better meets such requirements than Petrolagar?

To avoid bowel complications of pregnancy, Petrolagar is prescribed as a harmless routine.

Petrolagar has many advantages in maintaining bowel function. It is palatable and does not interfere with digestion. It produces normal, soft-formed fecal consistency, providing real comfort to bowel movement.

Petrolagar is an emulsion of 65% (by volume) mineral oil with the indigestible emulsifying agent, agar-agar.

Petrolagar

etrolagar Laboratories, Inc.,
536 Lake Shore Drive, SO-7
Chicago, Ill.

Gentlemen: — Send me copy of "HABIT TIME" (of bowel movement) and specimens of Petrolagar.

Dr.....

Address.....

.....

(Continued from page 44)

The twelve-room hospital being built at Crystal City by Dr. Cary A. Poindexter, formerly of Temple, has been completed. It will be open to all reputable physicians, either situated locally, or from adjacent towns or cities.

The John Sealy Hospital, Galveston, is expected to be ready for occupancy by August 1. Last fall a new power plant and laundry were erected at a cost of $250,000 to supply power to the new buildings as they were erected. This building is the first unit of the hospital program of the Sealy and Smith Foundation.

The Lubbock Sanatorium, Lubbock, has a capacity of eighty-five beds and fifteen bassinets. During the year 1929, 3,345 patients were admitted and ninety-three births recorded.

Deaths

Dr. Lewis Melvin Gooch, Paris, aged 36, died April 10.

Dr. Alexander Campbell Graham, Dallas, aged 84, died March 31, of lobar pneumonia and myocarditis.

Dr. James J. Hamilton, Eureka, aged 57, died March 8, of gangrenous cholecystitis.

Dr. William Dabney Lipscomb, Grapevine, aged 78, died April 8, of angina pectoris.

Dr. Titus Chauncy Loose, Dime Box, aged 56, died January 9, of heart disease.

Dr. William Kimbal Read, Texarkana, aged 58, died in March of heart disease.

VIRGINIA

Dr. Alfred L. Gray, Richmond, has been appointed Councilor of the Southern Medical Association from Virginia, the appointment having been made recently by the President of the Association, Dr. Hugh S. Cumming, Washington, D. C. Dr. Gray succeeds Dr. Lawrence T. Royster, University, whose term had expired.

Dr. Beverley Randolph Tucker, Richmond, was tendered a dinner by Dr. Howard R. Masters and Dr. Asa Shield, on the evening of April 26, as coincident anniversary of several important events in his life—his birth, the twenty-fifth anniversary of his graduation in medicine, and the fifteenth anniversary of the establishment of the Tucker Sanatorium at the present site.

It has been announced that a sum of $200,000 has become available for building and equipping a Nurses' Home at the University of Virginia, University, and that work will proceed at once.

Dr. R. H. Wright, Richmond, announces the association of Dr. Samuel P. Oast, recently of New York City. Dr. Oast will limit his practice to diseases of the eye.

Dr. Isaac A. Bigger, Associate Professor of Surgery at Vanderbilt University, Nashville, Tennessee, has accepted the professorship of surgery at the Medical College of Virginia, Richmond, effective August 15.

The Medical College of Virginia, Richmond, recently received a grant of $120,000, $40,000 from the Julius Rosenwald Fund and $80,000 from the General Education Board, for the construction of a dormitory and educational unit for the school of nursing of the St. Philip Hospital. This institution, owned by the College, is for negroes.

Plans have been completed for a four-story annex to Grace Hospital, Richmond, to cost approximately $75,000. The present officers of the Grace Hospital Corporation are: Dr. A. L. Herring, President; Dr. J. A. Rollings, Vice-President; Dr. T. B. Pearman, Secretary, and Dr. E. T. Trice, Treasurer.

The Medical Society of Virginia was represented at the U. S. Pharmacopoeial Convention, held in Washington, D. C., in May, by Dr. Alexander G. Brown, Jr., Richmond, Dr. J. C. Flippin, University, and Dr. P. W. Boyd, Winchester.

Dr. Herbert W. Lewis, Dumbarton, is taking special work in internal medicine and pediatrics at the New York Polyclinic School and Hospital.

Dr. B. F. Eckles, with Dr. W. A. Choate as his assistant, has assumed control of the Galax Hospital and Clinic at Galax.

(Continued on page 48)

ORGANOTHERAPY

can be effective only through the use of dependable endocrine products. The reputation and integrity of the manufacturer is the physician's only guarantee of reliability of those organotherapeutic products for which there is no chemical or biological assay. Every manufacturing process of all our products is supervised by our Analytical and Research Department.

DESSICATED PITUITARY BODY, U.S.P.
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Insure potency and constancy of action by prescribing the products of

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Urine actually becomes germicidal

Caprokol (Hexylresorcinol, S. & D.) is taken by mouth and excreted by the kidneys, appearing largely as a conjugate, but in sufficient concentration in the free state to impart active bactericidal properties to the urine.

Hence its activity in the treatment of urinary infections.

In addition, its marked analgesic action on the urinary mucosa brings prompt ease and comfort to the patient.

SOLUTION—*for children.*

CAPSULES—*for adults.*

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"A word fitly spoken—how good!"
This word came yesterday from a distinguished M.D.
—"The Storm has been tried and proven."

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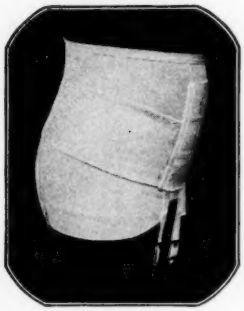

The New "Type N" Storm Supporter

meets demands of present styles in dress.

Long special laced back extension of soft material low on hips.

Hose supporters attached.

Takes Place of Corsets

Adaptable to Pregnancy, Ptosis, Hernia, Obesity, Sacro-Iliac Relaxation, High and Low Operations, etc.

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CLASSIFIED ADVERTISEMENTS

PHYSICIAN WANTED for a rural practice in Northern Virginia, fifty miles from Washington, D. C., in a rich, high, beautiful section with good schools, churches, excellent people and some bad roads. Collections 80 to 95 per cent—excellent territory, with good income. Please give the following information in your first letter: Age, married or single, number of children with ages; where born, where educated, when and where graduated in medicine; internship, if any, when and where; religion of self and wife; membership in fraternities, clubs, lodges, etc.; do you hold a license in Virginia? Address, Box 222, Warrenton, Virginia.

CHILDREN WANTED—Few younger children for excellent boarding school in Northern Virginia, near Washington, D. C. Girls not over twelve years old. Boys not over eight years old. Enroll now for session beginning September, 1930. Kindergarten. Regular courses, French, dancing, music. Apply to Dr. M. B. Hiden, P. O. Box 222, Warrenton, Va.

TECHNICIANS are in demand. We are successfully training laboratory and x-ray workers. Send us your assistant and we will return you a competent technician. If you are in immediate need of a well-trained helper, write or phone. Full information upon request. Alabama Pathological Laboratory, Birmingham General Hospital, Birmingham, Ala.

DRUG AND ALCOHOLIC PATIENTS are humanely and successfully treated in Glenwood Park Sanitarium, Greensboro, N. C.; reprints of articles mailed upon request. Address W. C. Ashworth, M.D., Owner, Greensboro, N. C.

(Continued from page 46)

Dr. H. H. Hurt has sold his stock in the Halcyon Hospital at South Boston, and entered on a Fellowship in Surgery at the Mayo Clinic, Rochester, Minnesota.

During the absence of Dr. Thomas G. Hardy on account of illness, the services of Dr. C. M. Nicholson, Charlotte Court House, were secured for the surgical work at the Southside Community Hospital, Farmville. Mr. E. Scott Martin is the newly elected President of the Hospital, for a term of two years, succeeding Dr. J. L. Jarman, who recently resigned.

Dr. and Mrs. I. K. Briggs have returned to South Boston from New York, where Dr. Briggs took a post-graduate course in surgery.

Dr. William F. Drewry, Director of the Bureau of Mental Hygiene, Richmond, recently addressed the Norfolk Branch of the American Association of University Women, at Norfolk, on the subject of mental hygiene.

Dr. Samuel Kendig Wallace and Miss Christine Tyson, Farmville, were recently married.

Dr. Barton Bates McCluer, Bon Air, and Miss Virginia Allen Chapin, Richmond, were married April 30.

Dr. John Staige Davis, Jr., University, and Miss Camilla Ruth Cole, New York, were married April 26.

Dr. Webster Parker Barnes and Miss Emmie Mae Marsh, both of Richmond, were married April 7.

Dr. James L. Hamner, Mannboro, and Miss Rebecca Sydnor, Marlinton, were married April 17.

Dr. Ernest C. Fisher and Miss Mary McCaw, both of Richmond, were married April 12.

Deaths

Dr. John Richard Adams, Blackstone, aged 83, died April 22.

Dr. Mary Evelyn Brydon, Richmond, aged 51, died April 13 of pneumonia after an illness of several weeks.

Dr. Benjamin W. Bohannan, Mathews, aged 72, died April 27.

Dr. Richard Terrell Davis, Fredericksburg, aged 58, died April 16.

Dr. Charles Duncan Garrett, Rockymount, aged 28, died March 17 of tuberculosis.

Dr. W. E. Oliver, Elliston, aged 75, died April 24.

Dr. Spurgeon John Railey, Handsom, aged 45, died March 26.

Dr. John A. Tyree, Danville, aged 43, died in March of nephritis.

Dr. Rawley Martin Witten, Graham, aged 76, died February 26.

WEST VIRGINIA

The West Virginia State Medical Association, at its recent annual meeting, elected Dr. C. H. Maxwell, Morgantown, President; Dr. Albert H. Hoge, Bluefield, First Vice-President; Dr. H. L. Goodman, Ronceverte, Second Vice-President; and Dr. T. M. Barber, Charleston, Treasurer.

Ohio County Medical Society has elected Dr. H. W. Bond, Wheeling, President; Dr. Robert J. Reed, Jr., Vice-President; Dr. H. C. Harpfer, Secretary; and Dr. R. W. Lukens, Treasurer, all of Wheeling.

Dr. W. T. Henshaw, State Health Commissioner, has announced that the next meeting of the Public Health Council for licensure will be held at Martinsburg on July 8-10.

A campaign to build a new hospital at Grafton has been started, the Grafton City Hospital being overcrowded and inadequate.

Dr. Joseph Lyon Miller, Thomas, was awarded the honorary degree of doctor of letters by the Medical College of Virginia, Richmond, on June 3. Dr. Miller is an alumnus of that College and is distinguished as a medical historian, bibliophile, and collector of rare medical books.

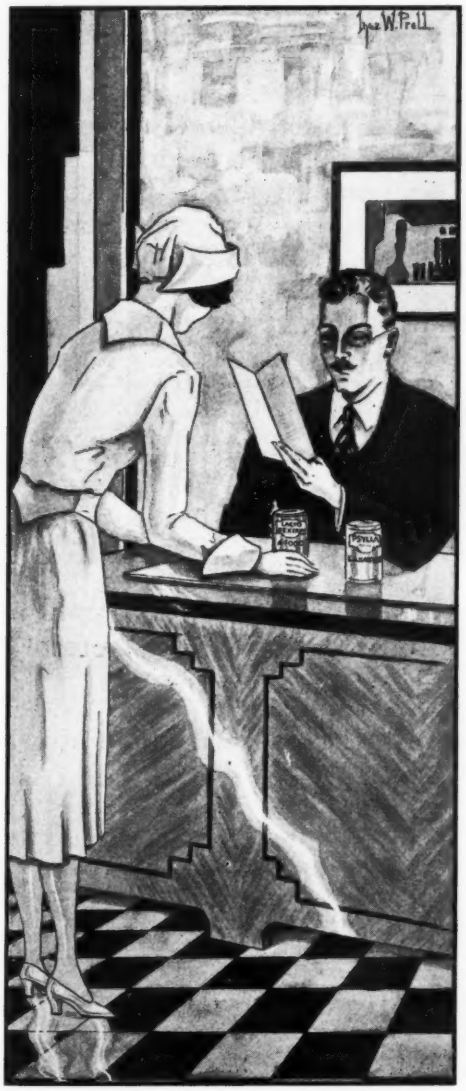
Dr. Robert W. Fisher and Mrs. Anna C. Jeffries, both of Morgantown, were married in Cumberland March 31.

Deaths

Dr. Frank Preston Beaumont, New Cumberland, aged 65 years, died May 8.

Dr. Walter Jacob Withers, Huntington, aged 50, died February 26, of pneumonia.

NORMAL FERMENTATION Combats Putrefaction



FOODS which ferment outside the body, ferment in the colon. Foods which putrefy in the pantry or the storeroom, will also putrefy in the colon.

To substitute a normal fermentation in the colon for a harmful putrefaction, carbohydrate is needed to enable the protective germs to gain the mastery.

Ordinary carbohydrates (starch and sugar) which form the bulk of our daily food do not answer the purpose because they are absorbed in the small intestine.

Only two carbohydrates have been found suited to this purpose—namely, Lactose and Dextrine.

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Lacto-Dextrin

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The coupon brings you a copy of this interesting and valuable book, together with a trial package of each.

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Send me, without obligation, trial packages of Lacto-Dextrin and Psylla; also copy of treatise "The Intestinal Flora."

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Dextri-Maltose for Modifying Lactic Acid Milk

In using lactic acid milk for feeding infants, physicians find Dextri-Maltose the carbohydrate of choice:

To begin with, Dextri-Maltose is a bacteriologically clean product, unattractive to flies, dirt, etc. It is dry, and easy to measure accurately.

Moreover, Dextri-Maltose is prepared primarily for infant-feeding purposes by a natural diastatic action.

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DEXTRI-MALTOSE NOS. 1, 2 AND 3, SUPPLIED IN 1-LB AND 5-LB. TINS AT DRUGGISTS. SAMPLES AND LITERATURE ON REQUEST. MEAD JOHNSON & CO., EVANSVILLE, IND., U.S.A.

In Rickets, Tetany and Osteomalacia—

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AMERICAN PIONEER STANDARDIZED ACTIVATED ERGOSTEROL

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Specify the American Pioneer Product—
MEAD'S Viosterol in Oil, 100 D
Mead Johnson & Co., Evansville, Indiana

"Hold Fast to That Which is Good"

The Mead Policy that for years has proved itself professionally and economically valuable to physicians who feed infants also applies to Mead's Viosterol in Oil, 100 D (originally Acterol). As with Dextri-Maltose, we feel that Mead's Viosterol is a part of the physician's armamentarium, to be prescribed by him alone. Therefore, we refrain from lay advertising of Mead's Viosterol or any other Mead Product; furthermore, we do not print dosage directions on the bottle, on the carton or in a circular. "Hold fast to that which is good" —the Mead Policy, Dextri-Maltose and Mead's Viosterol (originally called Acterol).

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MEAD'S INFANT DIET MATERIALS ARE ADVERTISED ONLY TO PHYSICIANS. NO FEEDING DIRECTIONS ACCOMPANY TRADE PACKAGES. INFORMATION IN REGARD TO FEEDING IS SUPPLIED TO THE MOTHER BY WRITTEN INSTRUCTIONS FROM HER DOCTOR WHO CHANGES THE FEEDINGS FROM TIME TO TIME TO MEET THE NUTRITIONAL REQUIREMENTS OF THE GROWING INFANT. LITERATURE IS FURNISHED ONLY TO PHYSICIANS.

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Whereas

The physicians of this country evidence an almost unanimous preference for Dextri-Maltose when modifying Protein Milk, as well as cow's milk formulae

Whereas

The cases requiring Protein Milk are difficult to feed, representing sick babies with severe nutritional upsets. Therefore,

Be it resolved

That, in the feeding of healthy babies, as a modifier of cow's milk, the physician's carbohydrate of choice is

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"DEXTRI-MALTOSE WITH VITAMIN B" IS NOW ALSO AVAILABLE TO PHYSICIANS WHO ARE INTERESTED IN ITS APPETITE-AND-GROWTH-STIMULATING PROPERTIES. PLEASE WRITE FOR SAMPLES TO MEAD JOHNSON & COMPANY, EVANSVILLE, INDIANA, U.S.A., SPECIALISTS IN INFANT DIET MATERIALS.

Mucous Membrane Inflammations

NEO-SILVOL is a valuable disinfectant in its specific field of treating mucous membrane inflammations without irritation. When the etiological factor is an infection—streptococcus, pneumococcus, staphylococcus, or gonococcus—solutions of NEO-SILVOL have been found dependable in soothing the inflammatory process, in controlling growth of bacteria, and in promoting a return to normal conditions.

NEO-SILVOL, a colloidal silver iodide compound, is effective without irritation. It does not precipitate tissue chlorides, or coagulate albumen, despite its antiseptic power. It leaves no disagreeable stains.

Select NEO-SILVOL for the treatment of any mucous membrane inflammation—in eye, ear, nose, throat, urethra or bladder.

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In 1-ounce and 4-ounce bottles of the granules.
In 6-grain capsules, bottles of 50, for making solutions.
As a 5% ointment in 1-drachm tubes.
In the form of Vaginal Suppositories, 5%—boxes of 12.

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